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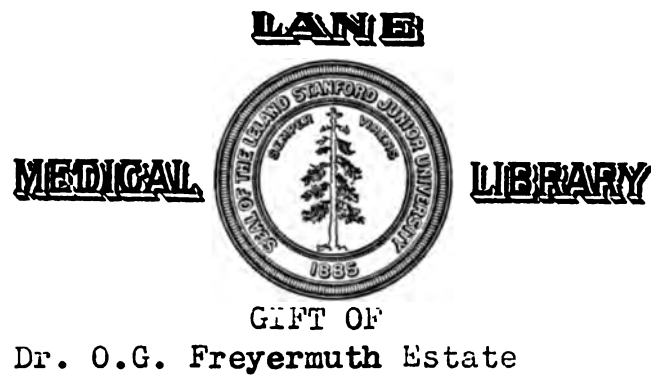
ANALYTICAL AND DIFFERENTIAL
DIAGNOSIS OF NERVOUS DISEASES
HENRY HUN, M.D.

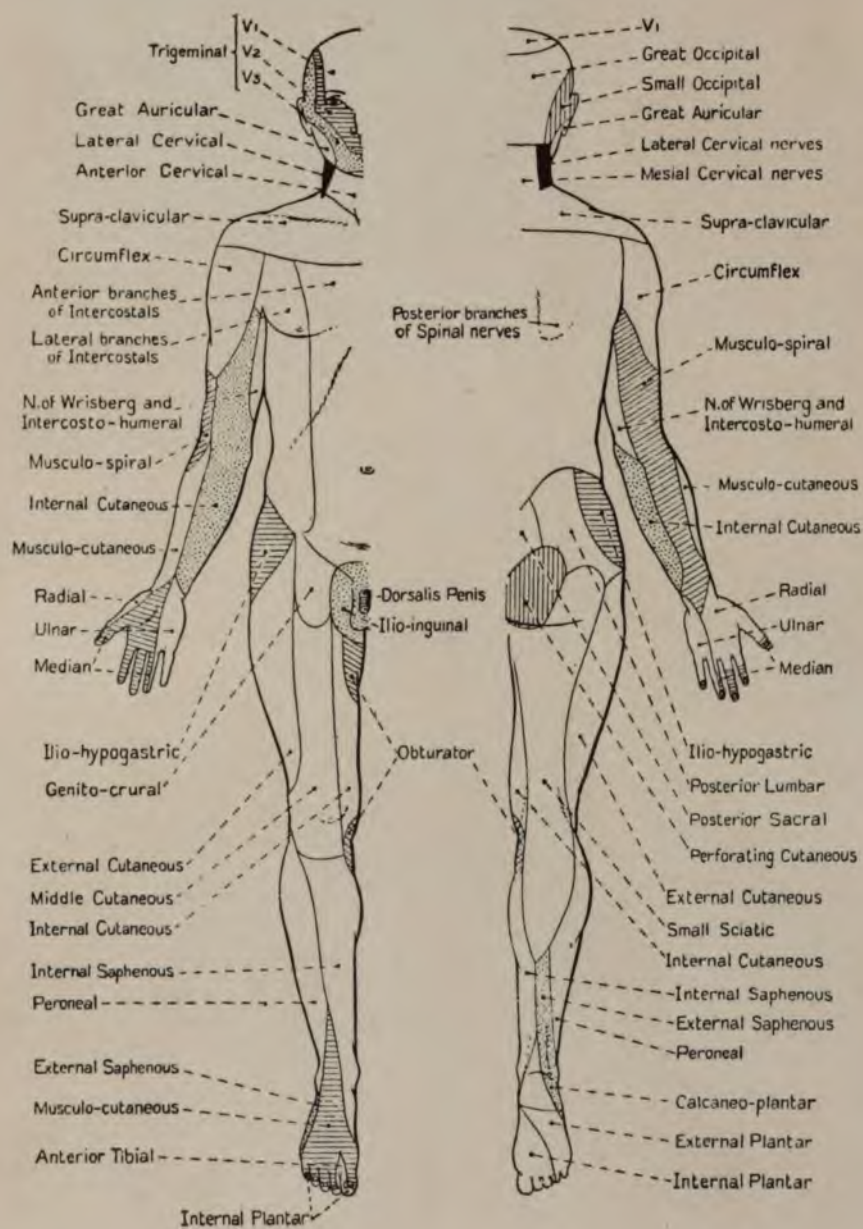


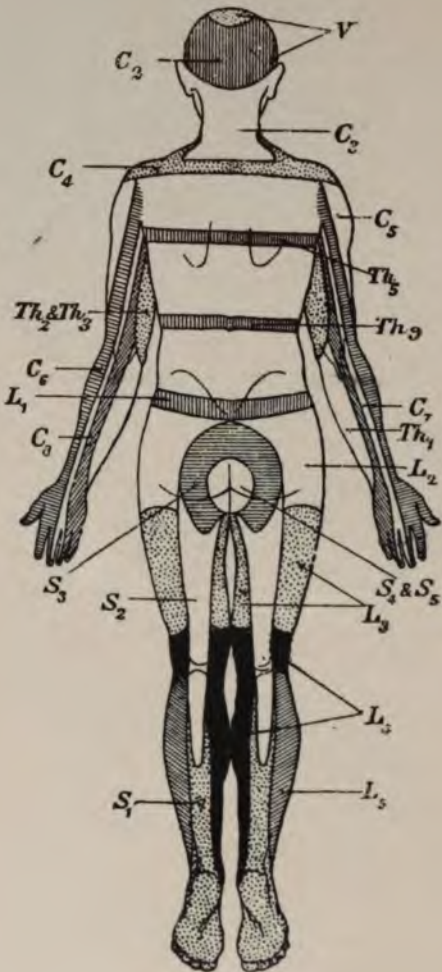
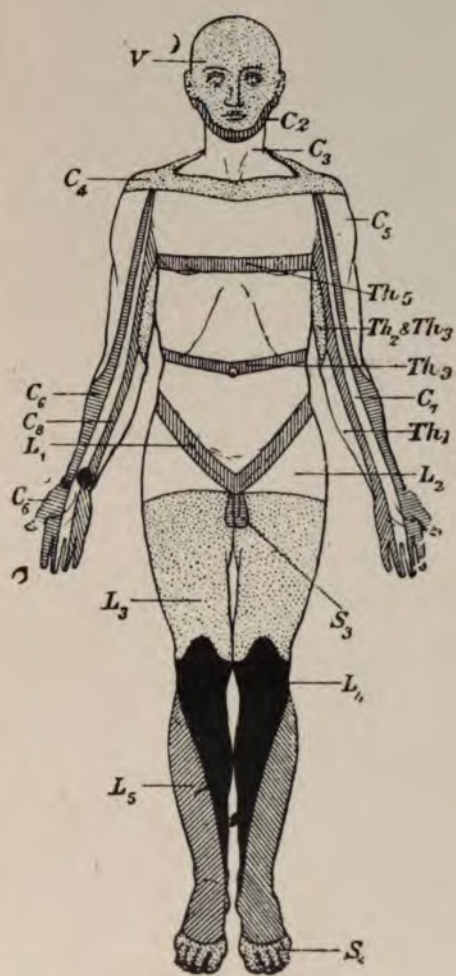
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AN ATLAS
OF THE
DIFFERENTIAL DIAGNOSIS
OF THE
DISEASES OF THE NERVOUS SYSTEM

ANALYTICAL AND SEMEIOLOGICAL
NEUROLOGICAL CHARTS

BY
HENRY HUN, M. D.

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*To
Thomas Hun
a loving father
a learned physician
a man of wisdom and wit
this book is dedicated
in most grateful remembrance*

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PREFACE

The diagnosis of diseases of the nervous system is generally regarded by medical students as one of the most difficult subjects in their course of study. It is so difficult that many students become discouraged and after a few attempts make no strong, continued effort to master it and, perhaps in consequence, physicians generally are weaker in this than in other phases of their work. In the hope of making this task less difficult for both physicians and students this book has been written. If the student can be taught to make the diagnosis of these diseases with comparative ease, it may happen that he will be led to undertake those further studies in the finer anatomy and physiology of the nervous system, which are essential for a full understanding of this difficult but fascinating department of medicine.

A careful physical examination and history of the case, as complete as can be obtained, are, of course, the essential basis of every diagnosis; but the commonly employed method of comparing the combination of symptoms thus obtained in any case with the various syndromes characteristic of the different diseases until a similar combination can be found, is not altogether satisfactory. More scientific and instructive is the analysis of each important symptom and the consequent ascertaining of the disease which must cause it under the circumstances (the other symptoms) existing in any individual case which may present itself.

In spite of its apparent complexity, the diagnosis of nervous diseases lends itself better than that of the diseases of most of the other organs to exact pathological analysis. Just as a chemist in analyzing a substance of unknown composition by a series of appropriate tests eliminates from consideration one group of chemical bodies after another until he finally discovers its class and name, so the neurologist subjects a patient to one test after another in definite sequence. As the result of each test he throws out of consideration one or more groups of diseases and assures himself that he has to do with a disease belonging to another definite group. With each successive test the number of diseases constituting a group becomes less, until finally one definite individual disease stands revealed among the few most closely related to it by a comparison of the remaining symptoms characteristic of each, which are given in the final abstracts. This analytical method is used, I think, by all great teachers of neurology in demonstrating cases of disease before their classes of students. It is the crystallization of this teaching into the tabular form which this book attempts to present.

In using this book for diagnostic purposes it is important that the "Introduction to the Diagnostic Charts" on page 119 should be carefully studied. By means of these charts it is possible to diagnosticate easily and rapidly any disease

of the nervous system and to localize the lesion, when any lesion exists. If the examiner makes a mistake at any point, the next step in the process or the abstract of the other symptoms of the disease will probably show him that he is in error and that it is necessary for him to retrace his steps.

For the sake of completeness certain trophic diseases are included, which, although causing a number of functional disturbances in the nervous system, are not really nervous diseases.

As might naturally be expected, the same disease, in so far as it presents many symptoms, appears a number of times in the different charts and even in the same chart; so that, in order to get a more complete idea of its symptomatology, it is essential that the different abstracts of it should all be read. To facilitate this, cross references by numbers within brackets are placed in the text.

Many diagnostic and technical terms are used which may not be familiar to the student; therefore these terms are classified, defined and their significance stated, as far as it is known to the author, in a series of semeiological charts preceding the diagnostic ones. Cross reference to these terms also is facilitated by the numbers within the brackets. A very full index, in the preparation of which the author has received much assistance from his friend, Dr. Dawes, also serves this same purpose.

The peculiar characteristic of this book on diagnosis is that it gives to the student or physician a key by which, in a comparatively easy manner from one or more important symptoms, he can arrive at a diagnosis. It also has the advantage that it divides the diseases into groups, the members of which have a definite relationship with each other; so that in the process of using the charts the student is constantly catching glimpses of the natural relationships between the different diseases of the nervous system. Although the symptoms of different diseases have often been contrasted in tables of parallel columns, in no other book, known to the author, has the subject been presented as it is here, and this must be his excuse for publishing it and for any defects which it may show, as there was no model which could be followed in preparing it.

In the preparation of the charts the author has received valuable suggestions and aid from several friends and especially from Drs. Mosher, Gordinier and Archambault, while for the plates he is greatly indebted to Drs. Streeter and Hawn. To these, his present friends and former students and assistants, he gratefully acknowledges his indebtedness and returns his thanks.

It is very gratifying to the author that the first edition of two thousand copies has been exhausted in eighteen months, giving him an opportunity of making a complete revision of the text, and of adding a few plates and also introductions both to the semeiological and to the diagnostic charts, which seem to him to add much to the value of the work.

The criticisms of Dr. Mosher and his untiring aid in putting both editions through the press are large factors in whatever success the book may have.

HENRY HUN.

Albany, N. Y.,
August 1, 1914.

PART I
SEMEIOLOGY
THE EXAMINATION OF PATIENTS
AND A
PHYSIOLOGICAL AND PATHOLOGICAL ANALYSIS
OF THE
RESULTS OBTAINED FROM SUCH EXAMINATION

AN ANALYSIS OF THE
SUBJECTIVE AND OBJECTIVE SYMPTOMS OF DISEASE

INTRODUCTION TO THE SEMEIOLOGICAL CHARTS

The diagnosis of nervous diseases, if it is to be at all satisfactory and accurate, must be based on anatomy and physiology. The practitioner is supposed to have some acquaintance with these subjects, and the curriculum of the medical college is so arranged that the student is taught them before he commences clinical work. It seems, however, desirable to make here an attempt to present a brief, but comprehensive, outline of the physiology of the nervous system, including some statements as to its anatomy, which latter can be supplemented by an inspection of the plates at the end of the book.

The fundamental element, or unit, of the nervous tissue is the *neuron* (461-4) a cell with many processes projecting from it; some short and branching (dendrons), one (rarely two) which often extends a long distance and usually becomes the axon of a medullated nerve fiber, and which, in some cases, gives off a few collateral branches. Both axons and dendrons are composed of delicate fibrillae which pass directly without interruption through the cell body. Of these neurons, varying in form and size and supported by the delicate framework of the neuroglia, the entire nervous system is composed.

The fundamental physiological characteristics of the nervous tissue are *excitability* and *transmission*: the power of receiving an excitation and transmitting it from one end of the neuron to the other and of transmitting it to other neurons with which the first is in anatomical and physiological relationship or contact. By its dendrons the nerve cell receives nervous impulses and by its axon it sends out its own impulses. There is experimental evidence which tends to prove that the activity of a nerve cell is the result of chemical reactions (consumption of chromatophilic substance, etc.), while the conduction along nerve fibers is mainly a physical process. The transmission of energy from one neuron to another in contact with it seems to depend upon differences in the tension of this energy in the two neurons. The cellular activity is, therefore, easily exhausted, while the activity of the nerve fiber is not easily exhausted.

Of the numerous forces and forms of energy in the world only a portion can be perceived by man. It is certain that some animals perceive things imperceptible to him. The various forms of energy in nature cannot act directly upon the nerve to produce sensory impulses, but intermediate organs, "end-organs," are necessary to transmute the external energy into nervous energy. In virtue of chemical changes the potential energy stored in the end-organ becomes active; the inciting cause of this being the external or foreign irritation. There are, doubtless, many forms of energy in the world which cannot be perceived, because there are no suitable end-organs to bring about this transmutation. Sometimes this can be accomplished by adding to the end-organs some mechanical contrivance suitable to bring about this transmutation; as for instance, the fluoroscopic screen for X-rays. The universal ether is doubtless in vibration far beyond the limits of about four hundred million million per second, which constitute for us the color red, and about seven hundred and sixty million million, which constitute violet; and indeed we have reason to believe that the ultra-violet rays have some effect upon our body, but beyond the above limits the vibrations of the ether are not recognizable by our eye and brain. The absence of a sensory end-organ limits the number of perceptions and consequently the content of consciousness, but this content is, or can be, much larger at the present time than in times past. Many new forces (X-ray, etc.) are now perceptible by the organs of sense which were before imperceptible. The sensory apparatus also is not absolutely perfect. Things moving very rapidly cannot be seen. The spokes of a rapidly revolving wheel cannot be seen.

THE ORIGIN AND TRANSMISSION OF SENSORY IMPULSES

Cutaneous and muscle-joint sensation (Chart VIa). The surface of the body and the cavities connected with it contain organs: the terminal organs of sense (the sensory "end-organs"), which bring the body into connection with some, but probably far from all, of the forces of nature, and which "end-organs" are so constructed as to transmute physical forces (light in the eye; sound in the ear; heat, cold, touch, pressure and pain in the skin, etc.) into nervous excitations in the terminal filaments of the peripheral nerves. The skin contains many of these isolated terminal sense organs and, therefore, sensibility is not spread uniformly over the skin, but is located in individual points. From these points of greatest sensibility, its acuteness diminishes concentrically. In every square centimeter of skin there are, on the average, 12 to 13 points for cold, 0 to 3 for heat, and 24 for pressure, impressions; although these figures vary very greatly for different parts of the skin, the points being most numerous on the finger tips and fewest on the back. Most observers maintain that there are distinct points also for painful sensibility. Where the skin is stretched over bone (the malleoli), sensation is less distinct and is more distinct where the skin is hairy; a point for tactile sensibility being situated at the base of most, if not of all, hairs. There may be a delay of several seconds in the conduction of painful impressions, and there may be a summation of painful impressions; so that with repeated pin-pricks the pain becomes more acute. After section or injury of a nerve, the anesthesia and analgesia are never so extensive as is the area of distribution of the nerve, and sensibility often returns before the regeneration of the nerve has taken place. This is partly due to peripheral anastomoses and partly to recurrent fibers of the sensory nerve.

Head and his colleagues, after much experimental work on the subject, arrived at the following conclusions:

There are in the peripheral areas three kinds of sensibility, due to there being three different kinds of nerve fibers supplied to each area.

1st. *Deep Sensibility*. Muscle sense, pressure sense, pressure pain and localizing sense. This sensibility is conveyed by sensory nerve fibers, more or less deeply situated, beneath the skin and usually running with the motor nerves. When the motor nerves of the muscle tendons are cut, these forms of sensibility are lost.

2nd. *Epicritic Sensibility*. Tactile sensibility for slight impressions, form and space sense, sense of moderate, not extreme, temperatures (22° to 40° C.) and the precise localization of pain and temperature sense. This area of sensibility is very constant for each individual nerve. The restitution of this form of sensibility is very slow and is not complete until after several years.

3rd. *Protopathic Sensibility*. Pain and sense of extremes of temperature (below 22° or above 40° C.). These symptoms are accompanied by paresthesiae and a false localization. This form of sensibility is best tested on the periphery of the affected area, where the anesthesia is not complete, or over the whole area while regeneration is taking place. The restitution of this form of sensibility is relatively rapid (7 to 10 weeks).

These researches of Head are of great interest and value and have attracted much attention and discussion, but they are not, in their entirety, accepted by all neurologists.

Sensory impulses of all kinds are carried to the central nervous organs by the sensory nerves. Of these, the spinal enter the cord through their cell bodies in the spinal ganglia and through the posterior nerve roots (Fig. 26); while the fibers of the trigeminal, the great cranial nerve supplying sensation to the face, after passing through their cell bodies in the Gasserian ganglion, enter the trigeminal sensory nucleus in the pons (Fig. 19). The fibers from the posterior nerve roots, on entering the spinal cord, are sorted according to their physiological function into three great parts (Fig. 26). One part, which conveys tactile, pressure and muscle-joint sense impressions, ascends, mainly without decussating, in the posterior columns to the nuclei of the columns of Goll and Burdach, and thence is continued by a new set of neurons (the internal arcuate fibers), which decussate and pass through the median lemniscus (Figs. 20-3) to the optic thalamus, whence it is continued, also by another set of neurons (relays), to the parietal cortex. The second part, which conveys impulses for co-ordination, passes to the cells of the column of Clarke and

thence, mainly without decussating, through the direct cerebellar tract in the outer part of the lateral column and through the restiform body to the cerebellum. The third part, which conveys temperature and painful impressions, passes through cells in the posterior horn, decussates in the central gray matter of the cord and passes upward in the antero-lateral column through the spino-thalamic tract and lateral portion of the formatio reticularis to the optic thalamus, and thence to the cortex.

A destructive lesion either in the terminal end-organ, or at any point of these sensory tracts or neurons, causes a corresponding paralysis of sensation (anesthesia); while a slight, irritative lesion may cause hyperesthesia, paresthesiae or pain in the distribution of the nerve.

In addition to the anesthesia, which occurs in organic disease of the nervous system, there is an anesthesia which occurs in hysteria: hysterical anesthesia. This hysterical anesthesia, occasionally but rarely, involves the organs of special sense. It more commonly involves cutaneous sensibility and then the anesthesia is not limited to the distribution either of a peripheral nerve or of a nerve root. It may instantaneously disappear. It may recur in the same place, or in some other locality. It does not prevent the use of the part in performing acts, in which sensibility is essential. Upon excitation of the anesthetic parts vascular reflexes occur, which is never the case in anesthesia due to organic disease, peripheral or spinal. It is evident that this hysterical anesthesia is the result of imagination or delusion. It is purely psychic.

Pain (374) is an unpleasant sensation which never occurs in health, but only when the body is injured, either mechanically or chemically. It is a signal of warning that the body needs protection. Its intensity depends not only upon the intensity of the mechanical or chemical irritation, but also upon the condition (inflammatory, etc.) of the peripheral nerves and of the cerebral cortex. It is more intense when accompanied by fear and apprehension. An unexpected wound is less painful than an anticipated one. Pain is often associated with the allied perceptions called "paresthesiae" (375) which at times precede, at times accompany, and at times follow, the pain, and which are usually of central origin and are due to irritation of the sensory fibers at some part of their course through the central nervous organs.

Gustatory sensation (Chart VIa). The mucous membrane of the mouth (in addition to the terminal organs for tactile, pressure, thermic, painful, etc., impressions) contains also the terminal organs of the nerves of taste: the taste buds or bulbs, so called from their form, embedded in the epithelium of the mucous membrane of the mouth, especially of the tongue. Excitation of these taste bulbs gives rise to four distinct gustatory sensations: sweet, acid, salty and bitter, to which may, perhaps, be added alkaline and metallic. Many so-called tastes are really a combination of gustatory and olfactory sensations. The nerve fibers arising from the taste bulbs on the posterior portion of the tongue pass by the glosso-pharyngeus nerve in a direct manner through the petrous ganglion to its nucleus in the medulla, whence they ascend with the other sensory fibers of the lemniscus to the optic thalamus, and thence to the cerebral cortex posterior to the gyrus hippocampi (Fig. 16); while the nerve fibers arising from the taste bulbs on the anterior portion of the tongue pass at first in the lingual nerve and, soon leaving this, form the chorda tympani, which joins the facial nerve and runs with it through the Fallopian canal to the geniculate ganglion. Here the fibers divide; a part continuing alongside the facial nerve and forming the nervus intermedius, which runs to a nucleus in the medulla close to the glosso-pharyngeal nucleus; while the rest of the fibers run through the petrosal nerves and join the fifth nerve and pass to the Gasserian ganglion (Fig. 36), and thence to the cerebral cortex (Fig. 16). A destructive lesion at any point of this course will cause unilateral loss of taste (ageusia). When the lesion is in the Fallopian canal, the ageusia may be associated with facial paralysis on the same side.

Olfactory sensation (Chart VIa). The mucous membrane of the nose, analogous to that of the mouth (in addition to the terminal organs for tactile, thermic, pressure, painful, impressions), contains also the terminal organs of the nerves of smell. The nerves terminating in these organs pass upward through the cribriform plate to the olfactory bulb, and thence backward through the olfactory tract: some to the anterior perforated space and sub-thalamic region

(olfactory reflexes) and some to the cortical center for smell in the gyrus hippocampi (olfactory perceptions) (Fig. 16). Each olfactory bulb is connected through the anterior commissure with both cortical centers.

Hearing (Chart VIa.) The terminal organ for hearing is the organ of Corti in the cochlea, within the petrous portion of the temporal bone. In this organ there is a long series of vibratory structures of unequal lengths; so that on them can be reproduced every possible tone with its over-tones or harmonics. The auditory nerves terminating in this organ pass to the ventral and dorsal auditory nuclei in the pons. From these nuclei fibers pass upward, some decussating and some not, through the lateral fillet to the corpora quadrigemina, and thence through the sub-lenticular region of the internal capsule, posterior to the fibers for cutaneous sensibility, and reach the cortical auditory center in the anterior part of the superior temporal convolution (Fig. 15). A destructive lesion of one auditory nerve will cause unilateral deafness on the same side, but a lesion of the tract connecting the sub-cortical with the cortical centers, since this tract contains both crossed and uncrossed fibers, will not cause any deafness. Deafness results only from a bilateral central lesion affecting the corpora quadrigemina or the sub-cortical tracts described above. Even destruction of the auditory cortical center in both hemispheres does not seem to cause complete deafness.

Sight (Chart VIa.) The terminal organs for sight are the rods and cones in the retina within the eyeball. The rods seem to be concerned in seeing in dim, the cones in bright, light. They vary in relative numbers in different animals, according as they roam by night or by day. From these structures start the terminal filaments of the optic nerves, which run backward from the eyeballs. In the optic chiasm the fibers from both maculae luteae and from the nasal half of each retina decussate; so that in the left optic tract are collected all the fibers from the left half of each retina (right visual field) and those from both maculae luteae; while in the right optic tract are collected all the fibers from the right half of each retina (left visual field) and those from both maculae luteae. The fibers of the optic tract on each side terminate in the external geniculate body, the pulvinar and the anterior quadrigeminal body of the same side, and are thence continued through the posterior portion of the internal capsule and the fasciculus of Gratiolet to the lips of the calcarine fissure on the median surface of the occipital lobe of the same side (Fig. 37).

A destructive lesion of the optic nerve causes blindness of the corresponding eye, but a lesion of any portion of the optic tract, or geniculate body, or fasciculus of Gratiolet, or of the lips of the calcarine fissure will cause homonymous hemianopia of the field of vision of the opposite side; while a lesion of the central portion of the optic chiasm will cause binasal hemianopia.

Internal or general sensation. In addition to these sensory impulses which convey to the brain excitations from the special sensory organs and hence from the external world, there are others which come from the different organs of the body and, in case they reach the cortex, give rise to what is called internal or general sensation. Some of these internal excitations remain entirely peripheral and affect mainly the blood vessels; others reach no further than the spinal cord or ganglia at the base of the brain and incite those automatic acts which preserve the nutrition and the life of the individual; while others reach the cerebral cortex and at times affect profoundly the processes taking place in it. These general sensations have much influence on a person's emotions, moods, thoughts and actions. Hunger may entirely alter the normal acts of a man or beast.

Such internal or general sensations are for the most part ill-defined and ill-localized. They seem to depend upon the blood supply and upon the activity of the different organs and upon the state of contraction of the hollow organs; and they appear to have much to do with our feeling of comfort or discomfort, which latter may amount to even severe pain. The sensation of hunger seems to be caused by contraction of the empty stomach, and the various colics by contraction of the circular muscular fibers of the intestine, the ureter or the bile duct. But the best understood of all these internal or general sensations is the composite one called muscle-joint sense, which is mainly made up of impulses from the muscle and its tendon and the articu-

lating surfaces and also from impulses from the skin and other tissues in the neighborhood, as these are stretched or relaxed in motions of the joint. The muscle-joint sensory conduction we have already considered in connection with the conduction of tactile sensory impulses. To these internal sensations must be added also, probably, the cortical innervation feelings (see later) inasmuch as a person often feels that he is moving, or feels that he knows the position of, a paralysed or even amputated limb.

SENSATION (CHART VI)

When these various impulses have passed along the various tracts and have traversed, and been interrupted by, several masses of gray matter, they reach the sensory area of the cerebral cortex and there give rise to a new form of energy called sensation. That is to say, a physical force (as for instance, ether in rapid undulation) is converted in a terminal organ into nervous energy, and as such, having traversed the sensory tracts, reaches the cerebral cortex. It is there transmuted into a new form of energy (as for instance the sensation of light). The sensation of light takes place in the brain, not in the eye, and has no similarity to the undulations of ether from which it normally originates, and it may, indeed, be caused not only by these, but also may originate, in perfect darkness, from mechanical irritation of the eye (as by pressure from the finger upon the eyeball) or of the optic nerve. Sensation is, therefore, rather a symbol than a picture of the external object, with which by experience it is associated.

Sensation is thus a special, individual force, similar to electricity, light, etc., which is produced in the cerebral cortex and which has its special, individual characteristics. A complex manifestation of this force constitutes consciousness and personality. Sensations originating from the different organs of sense are located, as we have seen, in different and special portions of the cortex (Figs. 15 and 16) and do not at all resemble the external phenomena causing them. A clap of thunder and a flash of lightning are very different external phenomena, but the reactions in the cortex, which constitutes these sensations, probably vary in topography rather than in quality. We know nothing more of the essence of this form of energy, which we call sensation, than we do of the essential nature of electricity, or of contractility in the muscle fiber or in the amoeba. We know something of its effects and we know something of the locality of the cerebral cortex in which it occurs (Figs. 15 and 16) and that it is very dependent upon abundant blood supply and that it must result from chemical actions taking place in the cortex. Conscious sensation, probably occurs only in those animals which possess cerebral hemispheres.

Sensation and all other forms of mental activity are absolutely dependent upon a fairly healthy cerebral cortex and a fairly abundant blood supply to it. When the cerebral hemispheres in an animal are removed, or when the cerebral cortex in man is entirely or mainly destroyed by disease, or in a child the hemispheres are absent or very defective, or when the blood supply is cut off from the cerebral cortex altogether or in large part, then sensation, perception, memory, thought, emotion (and its corporeal expression), ethics, association of ideas, voluntary motion, inhibition, intelligence, personality and consciousness are all lost.

Sensation is the simplest manifestation of consciousness (see later) or cognition. For its production a certain degree of intensity of the nervous impulses is essential, below this point of intensity the cortex may be in activity, but sensation will not result; the activity will be sub-conscious. A series of these slight impulses quickly repeated may by summation cause sensation. There is, therefore, a minimum of intensity necessary for sensation; just as electricity passing through a wire must have a certain intensity before the wire glows and light is produced. There is also a maximum beyond which, no matter how great the irritation, there is no increase of sensation, but rather a diminution from exhaustion of the nerve cells. Between this minimum and maximum point, sensibility increases, or diminishes, not continuously, but by little steps; a definite ratio to the stimulus (Weber's law).

Furthermore, a weak or moderately strong excitation may reach the cortex at a time when other portions of the cortex are in such strong excitation that this weak irritation may produce no sensation, but remain sub-conscious. The line between the conscious and the sub-conscious cannot be sharply drawn.

PERCEPTIONS AND CONCEPTS (CHART VI)

A perception consists of a combination of sensations, which are obtained from various sensory end-organs, but all of which proceed, usually simultaneously, from the same external object. A perception of an apple is composed of several sensory impulses: of visual sensations from the retina, representing the outline and markings and color of the apple; of muscle sensation from the ocular muscles, representing its distance from the eye, its position in relation to other objects and to some extent its form; of tactile sensations from the hand, representing its form, firmness and texture; and of gustatory sensations from the mouth, representing its taste. The various physico-chemical changes, thus set in activity in the cortex, combine to produce the full perception of the apple. For a full and complete perception, consisting as it does of so many elementary sensations, quite an appreciable time, or frequent repetition, is needed. The development of a perception is found by experiment to proceed from generalities to details. A combination of the full perceptions of many apples, each resembling and at the same time in some respects differing from the other, produces the idea or *concept* of an apple, with which is associated its written and spoken name and any other experiences or knowledge which have become associated during our life with apples. (See also under *Associations*.)

Perceptions occur in the cerebral cortex in immediate proximity to the cortical termination of the corresponding projection fibers. Each cortical center consists of a smaller portion, in which the projection fibers terminate and a larger portion, in which perceptions take place and in which their memories are stored. Thus, the optic fibers terminate in the lips of the calcarine fissure, while the rest of the median and convex surface of the occipital lobe is devoted to optical perceptions and memories (Figs. 15 and 16). When sensations only, but not perceptions, can occur, as when that portion of the center in which the projection fibers do not terminate is diseased, the condition is called in general *agnosia*. When there is a failure of tactile perceptions the condition is called *astereognosis*; in failure of optical perceptions *soul-blindness*, or *psychic blindness*, and in failure of auditory perceptions *soul-deafness*, or *psychic deafness*, or *auditory or sensory aphasia*. When that portion of the cortex in which the sensory fibers terminate is diseased, both perception and sensation are abolished.

EMOTIONS (CHART III)

Certain activities of the brain are accompanied by feelings of pleasure or discomfort or even pain, and usually also, if these feelings are moderately intense, by changes in the functional activity of the internal organs, especially in the circulatory and respiratory systems, but also in the alimentary canal and in the other viscera and glands of the body. These feelings are due in great part to the internal or general sensations. When the bodily functions are disordered we have a general feeling of discomfort and when all is working well we have a sense of buoyancy and exaltation; all moves smoothly without friction, as in a well oiled machine. These internal sensations, as was mentioned on a previous page, are ordinarily the dominant factor in our feelings and emotions and greatly influence also our voluntary actions, which for instance may be altogether different in a state of hunger from those in a state of satiety. Indeed the internal sensations, such as hunger, etc., are very often themselves the cause of extensive voluntary acts, which have for their aim relief from this sensation. As these internal sensations vary from time to time, our moods change, and perceptions, which at one time are pleasant, may at another time be unpleasant. Irrespective of our moods, however, some perceptions are almost always pleasant, others are not. Things which tend toward the preservation and health of one's self and his family are usually pleasant, and vice versa. Perceptions to which we have become accustomed are usually pleasant, and even unpleasant perceptions by frequent repetition at times become bearable and even pleasant.

Not a few perceptions are accompanied with relief of discomfort, as when hunger is assuaged, or we accomplish something desired or in some way contribute to our well-being or success and thus give pleasure; while other perceptions act in a contrary manner. These feelings of pleasure and pain may be due in part to the intensity of the sensation or perception, in part to heredity, as a result of evolution in case of objects desirable for the health of the body; but in greater part

to associations (see *Associations*) with previous similar perceptions, and in greatest part with the feeling of satisfaction or dissatisfaction with the result of one's actions.

Sensations of moderate intensity are usually pleasant; while sensations of very great intensity, which produce abnormally strong reactions in the nervous tissue, are usually unpleasant. Sensations originating from sets of vibrations having a simple ratio to each other are usually pleasant, while those which have a complicated ratio are usually unpleasant. Foods which nourished our ancestors usually taste good to us. The child probably acquires a taste for sweet things from the sugar in its mother's milk. Most of our pleasant and unpleasant sensations are the result of our education. They are, therefore, much more pronounced in adults, especially educated ones, than they are in children. A perception which is associated with, or followed by, pleasure or pain will always, or for a long time, as often as it occurs actually or in memory, be accompanied by a pleasant or painful emotion, whether the memory of the original pleasant or painful result associated with it is present in consciousness or not.

Mankind does not find itself in this world with all its needs and wants satisfied; on the contrary everyone must acquire food, clothes, habitation, warmth and a hundred other necessities. A man who sees his neighbor with something good, which he has not, desires it, or something similar. These wants and desires are the great, almost the only, incentives to voluntary action. When this voluntary action results in success we have in it our greatest pleasure and when it results in failure, our greatest unhappiness. All things connected with our success receive an associated emotion of happiness; while those things connected with our failure receive an associated emotion of grief.

In these various ways a certain number of our perceptions have associated with them an emotion (204), or tone, of pleasure or pain, greater or less, and a series of such emotions, or one long continued, will make us happy or unhappy for a considerable length of time and will constitute what we call our "mood."

In certain abnormal states of the cerebral cortex (exhaustion, circulatory irregularities, poison and other less well known disorders) the emotions become dissociated from the ideas with which they are normally associated; so that all cerebral activity is accompanied by one emotion; in some cases, sadness; in others, fear; in others, joy and in others apathy or absence of all emotion. An emotion is often so strong and so occupies the patient's consciousness that it is impossible, or nearly so, to attract his attention.

MEMORY (CHART III)

When perceptions take place, chemical changes are occurring in a definite portion of the cerebral cortex, which not only produce the perception, but also leave thereafter a permanent alteration in the cortex. The force derived from the chemical changes taking place in the cortex during an active perception may result in a structural, physical or chemical change in the nervous elements, or more likely in the storing in them of potential energy, which can be liberated and become actual later. Memories are dynamic changes in nerve cells and fibers which reduce the resistance to subsequent similar impressions or excitations. Certainly, a definite change is brought about which registers a permanent memory of the object perceived and subsequently this memory can be latent (sub-conscious), or active (conscious), from time to time. Consciousness, the actual perception of an object and its associated active memories (active attention), is a very exhausting, energy consuming process for the cerebral cortex. Sub-consciousness, the preservation of memories, not present in consciousness, is not exhausting to the cortex, even though the memories be preserved for many years.

In virtue of this change in the cortex, a memory of this perception always results from irritation of this altered cortex. This memory may be aroused, or enter into cognition, by the external force which originally caused it and, then, the object will be *recognized* (re-known), because the actual perception corresponds perfectly with its memory; or the memory may be aroused by way of those association fibers which it had previously set into activity. Memories become associated with each other in accordance with the relationship of the objects causing

them, as the result of our experiences with these objects. In perception, then, a trace of the cortical excitation remains in the cerebral cortex as a memory, in a sense analogous to the persistence and after image in the retina after strong excitation (looking for some time at a bright light).

These memories are, however, very different qualitatively from the original perceptions. The former have no actuality. Even though they may be at times very vivid, they never seem real to a normal personality.

The whole cortex of the brain is in great part a huge store-house of memories. These memories are grouped together; so that those which are derived from the same organ of sense lie together in the cortex. This localization of memories has been worked out with great care and is to a considerable extent known. It is shown in Figs. 15 and 16. A local cortical lesion may thus produce a loss of a group of allied memories.

All memories are sensory in character with exception of one doubtful group. This group consists of memories of so-called "innervation feelings." When a muscle is contracted the person to whom it belongs has a feeling of this contraction and can estimate its strength. This feeling is called an "innervation feeling" and its memory is stored away in the cortex of the anterior central convolution and of the neighborhood in front of it. This innervation feeling seems to be essential for the voluntary performance of the corresponding act. When, in consequence of a cortical brain lesion in the area in which these memories are stored, a person loses the power of performing certain acts, he often says, "I have *forgotten* how to do it." These innervation feelings and memories do not obtrude themselves strongly into our consciousness. They have rather to be sought for, but they usually can be observed, except in those actions which follow very rapidly upon the perception, or memory, causing them. There are many physiologists and psychologists, however, who question whether there are any so-called innervation feelings or memories in consciousness. However that may be, whether true innervation memories exist or not, the function of this cortical area is an actuality and whenever a portion of the motor cortex is sufficiently excited by a perception, or a memory, from the sensory cortex, a so-called voluntary, or association, action results. (See *Voluntary Movements*.)

ASSOCIATION

The essential physiological characteristics of nervous tissue are: first, its excitability, its reaction to stimulation by the discharge of nervous energy stored within it; and second, its transmissibility, this nervous energy, whenever produced, does not long remain localized, but tends to pass along nerve fibers, throughout its own neurons and to other neurons. The channels along which it will pass depend upon the anatomical arrangement of the fibers. In consequence of heredity and evolution, certain channels are easier for the passing of this nervous impulse than are others. This is especially true of certain reflexes present at birth, such as breathing, sucking, etc. Other channels are made easy later in life by the constant passage of impulses along them. The more frequently an association fiber is used the better conductor it becomes. The way that has once been traversed and that has often been traversed becomes the easiest way. It is the way of least resistance and it is a universal rule, whether it be a foot-path, or a conductor of electrical or of other force, or a nerve fiber or cell, that the way of least resistance is the easiest way: the way usually followed. When not used for a long time, like a deserted garden path, the channel may be obliterated and the association lost.

When a perception occurs, impulses radiate out along the association fibers from that portion of the cortex which produces it. If at the same time another perception, or a vivid memory of a perception received a moment before, takes place in another portion of the cortex, the association fibers connecting these two portions of the cortex, where perceptions are occurring, or have just occurred, being acted upon at both ends, will convey impulses to and from more readily than the other association fibers. The longer and more frequently the association fibers are traversed by these impulses the better conductors do they become and these two perceptions become more and more easily excited the one from the other. The activity

in the cortex does not long persist; so that when the associated idea is in consciousness, the original perception which awoke it is already, or soon will be, sub-conscious. Yet they are firmly associated together; so that whenever in the future one enters into activity it may excite the other. Thus, association between perceptions of the events and objects received simultaneously, or immediately before or after each other, are formed in a never-ending stream and the events and objects are considered as contemporaneous and often as related to each other. Subsequent experiences may verify and strengthen some of these associations and may disprove and unmake others. Associations with any one perception may be, and usually are, extremely numerous. There is also an association of words as well as of perceptions, and the associations of words have no necessary relationship to the associations of the objects which they represent. Associations may be at first very imperfect and very difficult to form, but with repetition and practice become easy. The work of a child in school is difficult until by repetition he has learned thoroughly his lesson. Then the recitation is easy. Addition, subtraction, etc., are at first performed slowly and with difficulty but later, in consequence of frequent repetition, rapidly and easily.

When a number of perceptions are produced which are very similar and yet show more or less individual variations, as for instance perceptions of men or dogs, from a comparison of them and of memories of others, more or less similar, a *concept* or *idea* of a man or a dog is formed, which includes all the individuals. From many examples of individual freedom of action, the abstract idea, or *abstraction*, of liberty is formed. A great many such abstract ideas are gradually formed and this process is facilitated by the use of language. But each idea is the result of experience: the result of a conglomeration or generalization of one or more perceptions and their associations, and, by the aid of language, is given a name. It has been said that "we can understand only so much of an abstraction as we know individual cases which sustain it." Thinking and reasoning are much simplified and made more rapid by the employment of these abstractions.

ETHICS

Inasmuch as the sensory and motor areas of the cortex are intimately connected together, some perceptions lead to voluntary action, which may result in pain, either directly as physical pain, or indirectly as mental pain, the result of punishment or condemnation; so that the action and the perception which led to it will become associated with these unpleasant sensations or perceptions, and these associated unpleasant sensations will tend to restrain further similar actions. Such acts, bringing with them a penalty, will be called wrong and there will gradually be formed a large number of associations which will be identified with the ideas of punishment and condemnation and which tend to prevent the performance of wrongful acts; just as another combination of associations which have become associated with pleasure, reward or praise, will be associated with good or right. A person's idea of what is right or wrong will depend upon his education, the result of experience and of teaching, and is the basis of emotions and ethics, and of that ill-defined function, the so-called *conscience*, and may evolve into very elaborate and very controlling feelings and habits of thought. According as education has developed one set of these associations rather than the other, a good or bad character, not from his own but from the community's standpoint, is formed. These ethical ideas can of course be imparted from one person to another by language and, indeed, frequently are so imparted, but such ethical ideas are rarely so firm and convincing as those obtained from experience.

CONCENTRATION AND ATTENTION

It seems to be a general law in the physiology of the nervous system that when there is a strong activity in one part, the activity of the rest of the nervous system is inhibited. Thus, reflex activity can be inhibited by strong pain; and the reflex activity of the spinal cord is more or less inhibited when the brain is in activity. In the brain itself, when a portion of the cortex or a group of nerve cells, is in activity, the activity of the other cortical areas, as well as that

of the lower centers, is inhibited. The stronger the local activity, the greater and more extensive will be the general inhibition, and the more this active portion will have a free and uninterrupted field. Naturally, consciousness remains limited to this strong activity for a long time. When an unusual or very vivid perception or idea is in consciousness it occupies the center of the stage. Consciousness is limited to this one vivid idea and its associations; so that milder activities occurring in the cortex at the same time, which should produce, ordinarily, perceptions and associations, remain sub-conscious. This phenomenon is called *concentration* and is a very important function in nervous physiology. When the cause of this concentration is a perception, in addition to this inhibitory influence, impulses from the active sensory cortex radiate to the motor cortex and out to the perceiving organ and cause a change in its musculature in the form of greater tension, tonicity, change in its position, etc., which local change heightens the power of the organ for the perception of stimuli. Concentration is only another name for attention and has been regarded as an effort, and an expression, of the will or will-power, but the primary and essential factor seems to be the cortical activity. (See *Will*.) Not infrequently the concentration is centered about an unpleasant idea, from the thought of which we vainly try to escape; yet it is forced upon our attention and we cannot free ourselves from it in spite of every effort of our so-called will. We are at times in a state of "expectant attention" in regard to some possible perception, which state we cannot prevent, try as we may.

REVERIE AND THOUGHT

The steady stream of perceptions originating from the excitation of the various sensory organs is constantly awakening associated memories, and these memories other associated memories, and so, while consciousness remains passive, an ever-varying series of memories, visions, day dreams, etc., flow by. But consciousness may be active, and just as cerebral activities may cause either action or inhibition in the sub-cortical centers, so the whole mass and content of consciousness may to a certain extent strengthen certain memories and weaken others. This action of consciousness, like other nervous actions, grows stronger by use. So that a trained, educated, intellectual man, is able to keep one set of memories present in consciousness (attention), to call up associated memories, to reject some, to keep others active, to compare them all together. This is called the *act of thinking or reasoning*. The process of thinking is thus independent of speech; although speech is essential to its clear expression and certainly facilitates it, especially in its deep and profound forms. The question of *attention* is one which seems to require a more or less external *will* to keep the cerebral activity limited to one subject. Attention is, however, in part a manifestation of the association of ideas. If many associations at the same time bring into strong consciousness the desirability of investigating some one perception, this idea which we may call "a" and which will have widespread associations, will bring into consciousness this perception to be investigated, which we may call "b" with its various associations. If one of these associations leads to others remote and unrelated, and away from "b", it will not go far before it will awaken some of the associations connected with "a," even the absence of "b" from consciousness will do this, and "a" will be brought into consciousness and through "a" the investigation will be brought back to "b" again.

When we have forgotten a name, we often cannot by an effort of "will," however strong, recall it. The desire for the name starts series after series of associations in some way related to the name, which finally bring it into consciousness. Or the attempt may fail and the desire may be unsatisfied at the time. Hours or days afterwards the name may enter consciousness by some chance association and be recognized as the desired name.

IMAGINATION: CREATIVE FACULTY

Although usually one perception calls up its associated memories and keeps repeating them, in recalling again and again past events; yet unrelated perceptions and ideas may be present simultaneously, or nearly so, in consciousness and may be artificially associated together so that by such combinations ideas and scenes may present themselves, which are not the result

of our experience; or our former experiences may be changed or modified out of all relationship with themselves. This is called imagination. It is the creative faculty which shows itself actively in prose and poetic works of the imagination, or passively in day dreams; in contradistinction to true facts or real history.

CONSCIOUSNESS (CHART III)

Consciousness consists, at any instant of time, of the then present perceptions and of those past memories which are directly or indirectly associated with these present perceptions and which have been awakened by them into activity. Consciousness is thus a form of energy resulting from activity of the cerebral cortex. The other memories, not at that instant active, constitute sub-consciousness and may at any time become conscious memories. The content of consciousness embraces only a small fraction of those activities which take place in the brain and indeed only a fraction of those activities which take place in the cerebral cortex. The activity within the cerebral cortex must reach a certain intensity in order to produce sufficient energy to constitute consciousness. When this cortical activity, although existent, is less intense, we call the result of this activity sub-consciousness. Such sub-conscious activity may leave an ill-defined memory as the result of its action. The conscious and sub-conscious content of the brain together constitute a *personality*.

A new born babe has, probably, no consciousness. An infant attains consciousness slowly as he gradually obtains perceptions and memories and forms a large number of associations of all kinds. When an infant has his first perception, this one perception, together with certain rudimentary sensations he may have acquired, constitutes his entire consciousness and his entire intelligence. It is all he knows. As other perceptions are obtained and associated together his intelligence and his consciousness become larger, more distinct and more complete. The child in his development increases with great rapidity the number of his perceptions, less rapidly and subject to many subsequent corrections, that of his associations, still less rapidly his concepts, even more slowly his ethical and aesthetical ideas, and yet more slowly his abstractions; but at the end of a few years any perception or memory suggesting action is subjected to the interplay of all these activities before the action is done or left undone. In other words, consciousness and the act of thinking and reasoning on which action is based (see *Voluntary Motion*) are already, even in childhood, very complicated, perhaps more so than later in life, when action is mainly determined by habits of thought: by judgments firmly established by numerous experiences.

Consciousness is a form of energy or force, such as are light and electricity, which is transmuted from chemical action taking place in the cerebral cortex. Consciousness embraces all these chemical activities which have a certain intensity; below this degree of intensity these activities constitute sub-consciousness. The process is somewhat analogous to that in an electric-light bulb through which an electric current may be constantly flowing, but which only gives forth light when the current has attained a certain intensity.

Consciousness is constantly being newly formed and is dependent upon the perceptions, memories, feelings and ideas, ethical and others, present at any one instant. These phenomena themselves constitute and are consciousness. None of them, usually, continues long in consciousness. Others are constantly forcing them out. None remains constant. The continuity of consciousness is preserved by the mingling of memories of past perceptions with present ones, and by memories of past states of consciousness.

Consciousness is an active process and depends upon the integrity and the blood supply of the cerebral cortex. This blood supply is in constant ebb and flow throughout the different areas of the cortex; being at any instant most abundant in those areas which are in activity. Consciousness is a condition which, as yet certainly, we do not understand, although in a general way we regard it as the result of chemical changes taking place within the cerebral cortex. *The chemical changes themselves are not consciousness*, but they produce this form of nervous energy very much as a steam boiler and a dynamo, or a galvanic battery, produce electricity. We

are as ignorant of the exact nature of consciousness as we are of that of electricity. These chemical changes produce consciousness; a form of nervous energy; just as the chemical changes taking place in the muscles produce muscular force; a form of mechanical energy; just as chemical changes taking place in any living cell or tissue of the body produce a form of energy peculiar to itself. Consciousness thus locally produced, and thus continually produced, in the brain, passing according to definite channels to other regions of the cortex, surges through the brain, as memories and ideas are awakened and cause action and reaction. The subject is a most difficult one and is made, in a sense, more difficult by the faculty of language, which allows us to replace an idea by a word of somewhat uncertain definition and thus leads to uncertain and faulty reasoning, or to a high sounding sentence which means nothing. Consciousness, or cognition, seems to be something added on to the essential processes taking place in the brain. The various association-reflexes occurring in the brain could take place and do take place quite as accurately without consciousness, as for instance in the automatic acts of the somnambulist, or in the epileptic trance.

The brain is very abundantly supplied with blood, especially the cortex, and the latter is very sensitive to any interference with its blood supply. Loss of consciousness, which occurs normally in sleep and pathologically in many conditions, is caused much more frequently by a change in the quantity (anemia) or quality (drugs and poisons, including sepsis and other autogenetic toxic products) of the blood supply than by all other conditions combined. Perversions of consciousness, on the other hand, seem to depend less upon the quantity of the blood supply than upon its quality (poisons) and upon changes, organic or functional, in the cerebral cortex, especially upon its exhaustion.

PERSONALITY (CHART III)

Personality is the sum of the conscious and sub-conscious content of the brain. It expands as this content grows larger and better systematized. It becomes greater as during life a larger stock of energy is accumulated in its associated memories. It is stronger as the consciousness is more intense (virility). The "Ego" is the result of a long series of experiences (former perceptions) by which the body is differentiated from the external world (corporeal ego), and by which the complex of memories and ideas which the cortex has accumulated is differentiated from that of other individuals and is peculiar to itself (mental ego).

The totality of one's memories constitutes his experiences. Many similar memories, or experiences, are gradually combined into a general idea or principle which becomes a guide, or association channel, for future judgments and actions and may persist after the individual memories or experiences upon which it was founded have been lost. Cortical excitations of this nature are followed at once by actions which are almost involuntary (habits) and are not the result of a balancing of many former memories and ideas (thought). In this way one's character or personality is built up. Ideas firmly fixed by tradition, education and habit, acquire an overwhelming emotional value. They not only exist in spite of experience but even mould experience into conformity with themselves. Personality is the result of the manifold working of natural forces. Had the natural forces been different the personality would have been different. Each personality has its own history founded on its own personal experiences. *A man's personality has been created during and by his life, in a brain whose organization and capacity have been modified by heredity.*

Personality, being founded on, and consisting of, personal experiences, is strongly individual; but inasmuch as the large majority of men in the same community have very much the same experiences, and as they discuss these experiences with each other, there springs up between them a friendly feeling as beings of the same nature and with the same interests, needs, desires and aims. According to its education from its experiences a personality may keep itself apart from others and strive only for its own well-being and may thus be selfish (an egotist), or it may merge itself into the social life of the community and strive for the well-being of its fellow men as well as its own and thus be generous (an altruist). In spite of much in common,

each personality differs from others. Some by their educational experiences become contemplative men, others close observers, others men of action, etc. Some men are of weak character, who have always been indulged and have always followed the path of least resistance; some are of strong character, who have had to endure privation and have learned to control their desires. These different kinds of men cannot by any effort of will change suddenly their character, which has been formed slowly by countless past experiences, acting upon a brain the anatomical structure and physiological activity of which has been modified by heredity. The personality of a child has the potentiality of developing in the future, but the kind of development depends more upon the kind of future that is before it, than upon its heredity.

Personality seems to be the energy resulting from chemical changes which have taken place and are taking place in the cerebral cortex and to depend absolutely upon the integrity of the latter. When the cortex is exhausted, or diseased, personality may be changed under some unusual experience, resulting from the undue dominance of some local excitation of the cortex, either permanently or temporarily (double personality), or it may become completely lost (automatism).

INTELLIGENCE (CHART III)

Intelligence consists of the content of object consciousness. It is absolutely dependent upon memory, without which it cannot exist. The greater the number of memories and the more perfect and easier the recalling of associations, the greater is the intelligence. A person's memories depend primarily on perceptions derived from his sensory organs and on his experiences. The greater the number and the variety of his experiences the greater will be his intelligence, other things being equal. If any one sensory organ is absent or diseased from birth, memories of this sense will not be present and the intelligence will be diminished, unless this defect is in some way compensated for.

An increase of intelligence, though unusual, is not abnormal. In some cases this increase is due to a greater number of perceptions and ideas (the learned man); in some cases to better and wider associations throughout the entire sensory cortex (the wise man), and in some cases one portion of the cortex is functionally developed at the expense of others (the genius).

A diminution of intelligence may be due to imperfect development, to impaired nutrition or to destructive lesions of the cortex.

Perversions of intelligence, although they may, in part, be caused by peripheral lesions, are fundamentally due to disease, or poisoning, or malnutrition of the cerebral cortex.

SOUL AND MIND

All of these physiological activities of the cerebral cortex, which we have considered and which are popularly called "mental" or "spiritual," although they depend upon an inherited anatomical structure of the brain, are not present at birth. They are acquired, or created, during life by experience and by education in its broadest sense. They are the result of physiologico-chemical activity within the cerebral cortex. There is no scientific or trustworthy evidence of the existence of any further factor in the form of any ethereal essence, a "mind" or "soul" as distinguished from what has been described above as "personality." Indeed this assumption rather complicates than simplifies the matter, inasmuch as it is contradictory to one of the most firmly established principles of natural philosophy: "the law of the conservation of energy." This law has not only been established by irrefutable proof in the inorganic world, but also has been proved by experiment to be valid in animals and even in man. If the soul can produce or stop a cerebral activity of its own volition, thus creating or annihilating force which normally proceeds in an endless chain from one manifestation of force to another, then the law of the conservation of energy is no longer valid.

Mankind has been unwilling to allow that the causalities and laws, which prevail in physical activities, can be potent also in the body and still less in the brain and "mind," because this controverts all their preconceived notions of the soul and its relation to God. The prejudices (pre-judgments) of most men will not permit them to regard the mental activities as the

result of the physiological activity of the cerebral cortex, in the same way that the physiological activities of the other internal organs of the body produce and cause the functions of these organs. They are unwilling to regard psychology as identical with the physiology of the cerebral cortex. In earlier historic times, the winds from the cardinal points of the compass, rippling streams, cascades, the waves of the sea, growing trees and shrubs, etc., were each supposed to be animated by an indwelling spirit. At the present time, Naiads, Nereids, Dryads and other Nymphs: charming creatures of the imagination, have all been banished by the advance of knowledge. Only the indwelling, animating spirit of man: the soul, remains.

Whether an individual believes in a soul or not, depends upon his traditions, his education, his experiences and upon the personality which has been gradually created and developed during his lifetime by the combination of all those forces constituting consciousness and sub-consciousness, and which may, in a sense, be likened to the older conception of a soul. It seems probable that consciousness, intelligence, personality, etc., are forms of energy and force and may, perhaps, be called *spiritual* in contradistinction to *material*. Whether this energy or force, which is gradually accumulated during life, persists after death, we do not know. It certainly is absolutely dependent upon the blood supply of the cortex, and when this is arrested, personality with every other manifestation of consciousness ceases; but it still exists potentially and may be reanimated, if the circulation be restored after an interval of only a few minutes. If, however, the arrest of the circulation is so long that the cortex begins to degenerate or die, the personality is lost permanently, both actively and potentially.

Our knowledge in comparison with the wonders of the Universe is infinitely small. We know the relation of things, not their essence. But our knowledge is increasing and it is to be hoped that our children's children may have a higher point of view and a clearer vision.

INSANITY (CHARTS III AND XVI)

Whether we believe in the existence of a soul within the body or not, certainly insanity is no longer regarded as the possession of the body by an evil spirit (demoniacal possession). This was a well established belief for ages, but it has long since been abandoned and we now regard insanity as caused by abnormal cerebral action. The control of the body by a spirit, which we have finally rejected as regards insanity, the vast majority of mankind still retains for the healthy body, possibly because most of the few men who really think have not studied cerebral physiology.

We have considered briefly the actions taking place in the sensory area of the normal, healthy, cerebral cortex. In an abnormal cortex these actions are deranged. Local disordered cortical function produces local paralysis or apraxia or convulsions or even hallucinations; while general disordered function produces coma, neurasthenia, or insanity. Abnormal structure, whether the alteration be slight or great, and consequently abnormal function, of the cerebral cortex may be either congenital or acquired. The congenital form may be manifest in early infancy or may become apparent at any stage of the individual's development, as he successively meets tasks which require more and more intellectual power, when it becomes evident that his intelligence and ethics fall below the commonly accepted standards of the race or community of which he is a member.

The greatest degree of absence of intelligence is *idiocy* (1081), which shows itself almost at birth. In this disease the brain is so functionally incapable that it cannot produce perceptions, or register memories, or form associations, except of the most rudimentary kind. These persons, then, have no material for intelligence, consciousness or ethics and are incapable of speech. Next to this extreme degree is *imbecility* (1088), which may show itself at any time from birth to early childhood. In this class, simple perceptions, memories, associations and speech are possible, but only very imperfectly, and there is very limited material for consciousness, intelligence or ethics. A still slighter degree of this condition is only manifested when a considerable degree of intelligence or ethics is imperatively required, especially at the so-called critical periods of life, as at puberty (some forms of adolescent insanity—1096). Although individuals of this

class have memories, associations and consciousness, their intelligence, ethics and judgment are found to be inferior to those of their fellows educated under the same conditions and they are called *feeble-minded* and *defectives* (1092) in varying degree. In the slighter forms of this class only the higher and more complicated ideas, such as altruism and morality, are absent or impaired (moral insanity and some criminals). In the severer forms, the loss is more profound and involves all the cortical functions.

In other cases of congenitally defective brain the defect is very slight, but some of the association channels are more patent than others, and than is normal; so that certain associations and ideas are constantly being presented to consciousness and are called up by all kinds of unrelated associations and cannot be corrected, and consequently the cortical actions are distorted and twisted and irrational. To this class belong the *paranoiacs* (1113).

Education and training have much to do with the development of the activity of the cerebral cortex and consequently with the individual's intelligence and ethics. There are individuals who, partly in consequence of a defective brain, but mainly in consequence of a defective training and education, do not have normal experiences and form a number of abnormal associations and ideas, especially ethical. Such individuals comprise the majority of criminals and cranks. Such cases bridge over the separation between the congenital and the acquired forms of insanity. Of course, it is possible that a person with a normal brain, who is isolated from his fellow beings and receives no training or education, will be feeble-minded or even an imbecile.

All these congenital forms of insanity may be broadly classed under the term *amentia* (211, 1076) *in its widest sense*: the mind never having fully developed. On the other hand all the acquired forms of insanity may be broadly classed under the term *dementia* (212, 1077) *in its widest sense*: since there is always present a certain mental weakness not previously present, a falling off in greater or lesser degree from the previous more perfect cortical activity.

Acquired, disordered activity of the cerebral cortex resulting in insanity primarily also depends upon a defective brain, either hereditary or acquired, but secondarily upon many inciting causes. It may be due to a general deficiency in the blood supply consequent upon atherosclerotic arteries, as in *senile dementia* (1105); or may be due to an irregular cortical circulation consequent upon chronic meningitis; the most striking example of this class being *paresis* (1104). Various poisons (alcohol, etc.), endogenous or exogenous, are responsible for other forms of insanity which are usually, but not always, of comparatively short duration. Local lesions of the brain, such as abscess, tumors, etc., may in some cases alter the circulation of blood through the cortex generally and thus cause insanity. Exhaustion of the cerebral cortex from worry, anxiety, shock and other causes may cause insanity in persons with an unstable brain, as may also an anemic and altered condition of the blood. It is, of course, possible that several of the above causes act simultaneously, or in sequence, and as a matter of fact they frequently do so.

In most of the forms of insanity the altered cortical activity manifests itself in certain striking and unusual phenomena. One of these is hallucinations (213, 1078), which are abnormal perceptions. In hallucinations the symbol occurring in the cortex does not correspond to any external phenomenon, but is purely subjective, and is due to disturbances in the cortex itself, not in the peripheral sense organs. The hallucinations may occur in any of the special sensory regions of the cortex and hence may be either olfactory, gustatory, visual, auditory, tactile, or even visceral. The process in the brain which produces an hallucination must be similar to that which produces a perception. An hallucination is much more vivid than a memory and an hallucination is not a complete and correct reproduction of a former memory, but usually is something strange and bizarre. In some cases the hallucinations do not have the vividness of true perceptions, but seem to be internal voices of suggestions, telephonic communications or electrical action, etc.

An illusion (214) is also a false perception, but it originates from an external reality which is misinterpreted in the brain: the symbol in the cerebral cortex is not such as is usually associated by the average man with the external object, but rather a symbol usually associated

with a quite different object. Hallucinations and illusions may occur as the result of a local disturbance in a brain which may not be for the moment entirely normal, although the individual is certainly not insane. In such cases, hallucinations and illusions can be quickly dispelled by reason and by proof of their abnormal character. Insane persons, however, in consequence of a diffuse cortical disturbance, cling to their hallucinations and illusions with great tenacity in spite of strong proof to the contrary. These hallucinations and illusions occurring in a brain weakened by nature, poison or disease, naturally lead to abnormal associations and consequently to abnormal ideas. Abnormal associations will result not only from the strength and vividness of these hallucinations, but also because from patches of meningitis, or other cause, some areas of the cortex have more blood than others and, therefore, respond more readily to association impulses, near and remote. Moreover these abnormal ideas entering into consciousness and coming into conflict with former long established ideas lead to a condition of consciousness which we call bewilderment, clouded, befogged, confusion, distrust, apprehension, fear, etc. In these cases, impulses reaching the cortex normally from the organs of sense are so much weaker than the excitations already there, that they cannot enter into consciousness, but remain sub-conscious. They may, although sub-conscious, be registered and may be recalled to consciousness after the attack of insanity is past, but they have no present value and are inadequate to correct the abnormal activities and no sane judgment can result.

A cortex in which normal perceptions can occur only imperfectly, or not at all, and in which abnormal perceptions, associations and ideas are dominant, will naturally produce abnormal association reflexes, or actions. The simplest of these is *delirium* (217, 1107-8), in which the patient responds by word and act to the many false perceptions and ideas in his clouded and weakened consciousness. When the intensity of the process is less the false perceptions and ideas will produce *delusions* (215, 1079), which will cause abnormal and often dangerous association reflexes or acts. These delusions may remain isolated, unsystematized or may be woven in with all the real experiences of the individual life; so that a systematized delusion, founded upon more or less evidence or reasoning, acting upon a weakened or limited general cortical activity or judgment, results.

Often in justifying or explaining a delusion a patient will give reasons or cite experiences which we call false, but which are doubtless experiences, the symbols of which have occurred in his abnormally acting cerebral cortex. These delusions, or false and uncorrectable judgments, naturally lead to acts which are incompatible with an unconstrained life in a reasonable community. Naturally with all these abnormal cortical activities not only the ethical ideas of the individual are changed, but also the normal emotions associated with normal cortical activity are profoundly altered, whether in the form of exaltation or depression, either continuously or in alternation with each other.

In all forms of insanity, in consequence of its abnormal content, consciousness is altered and personality may be changed. There may be a double personality or the individual may imagine that he is dead, an animal, a king, or God, or, in extreme degrees of dementia, the patient may show no consciousness or personality at all. The emotions are also altered (morbid temperaments) in accordance with the ideas in consciousness, or may be entirely dissociated from the ideas with which they are normally in harmony, or may be entirely absent in extreme dementia, or may be feebly carried over from former highly emotional states. The emotion most frequently present, especially in the early stages of the disease is fear (phobias).

Fear and apprehension are prominent, even dominant, symptoms in the early stages of almost every case of insanity. The unusual, often monstrous, phenomena occurring in the cerebral cortex are so different from those previously present and so out of harmony with former memories and ideas, that the patients naturally become distrustful, apprehensive and full of fear. Many can hardly believe the information supplied by their own senses, much less the words of their friends. Some regard themselves as persecuted and as the victims of conspiracies. Fear is the dominant emotion within them.

The association reflexes are always altered in insanity in consequence of the abnormal cortical activity. In extreme dementia, voluntary motion is completely abolished. In profound melancholia, voluntary acts, as well as thought, are inhibited; while the reverse is true in mania, in which cortical activity, although abnormal, is greatly exaggerated. In consequence of prominent, compulsory ideas, so frequent in insanity, compulsory acts result.

MOVEMENT (CHARTS IV AND V)

When a sensory surface is irritated the animal often responds immediately by a comparatively simple movement, or the movement may occur only after a considerable space of time and may be very complicated, or it may never occur. Movements may also occur spontaneously, apparently not being preceded by any sensory irritation in the immediate past; although on careful analysis these spontaneous movements can always be referred back, indirectly, to some sensory irritation. All these different kinds of movements are divided into three great classes: reflex, voluntary and automatic.

REFLEX ACTION AND INHIBITION (CHART V)

A reflex act is a reaction from an irritation, which under like conditions always takes place in exactly the same way; it seems purely mechanical, as if a machine were working. The irritation may be a usual (normal or adequate), or an unusual (abnormal or inadequate), one; the former being much more effective, and it may affect the skin, mucous membrane, or muscle, tendon or fascia, or any of the organs of special sense. The impulse starts in the end-organ of a sensory nerve supplied to the sensory organ affected and passes centripetally along the peripheral sensory nerve fiber, or fibers, to the spinal or cranial ganglion, situated on the posterior spinal, or the cranial, nerve root. Thence it passes forward through the posterior horn to the anterior horn of the spinal cord, or through the brain stem to a cranial motor nucleus, and thence along a motor nerve root and peripheral nerve to a muscle or muscles, striated or unstriated, in which it causes a contraction, or to a gland in which it causes an alteration in its secretion (Fig. 24). Muscular tonicity is a variety of reflex action (240).

The various nervous elements traversed by the nervous impulses, as just described, constitute what is called "the reflex arc" (296). Slight irritative lesions of the reflex arc cause exaggeration, while destructive lesions cause abolition, of reflex action. This is the simplest form of nervous reaction and this is the simplest expression of it. Such simple reflex acts are the only ones occurring in the body during the early months of life and are unconscious acts. Similar reflex acts cause the respiratory and cardiac movements, the flow of saliva and other secretions, the vascularity of organs and the warmth of the body, and in general regulate the physiological actions of the body.

When the nervous impulse reaches the gray matter of the central nervous organs so many ways of transmission are open to it that it can pass by longer or shorter arcs or by several of them. The shortest possible reflex arc is through a peripheral ganglion (as in the vaso-motor reflexes). The next shortest is through the spinal cord. A longer one is through the spinal cord and the ganglia at the base of the brain. The longest is through the cerebral cortex. Some reflex acts, such as the pupillary (302), remain unconscious acts throughout life. On the other hand, most of the sensory impulses described above, when they have traversed the sensory nerves and have reached the sensory ganglia, pass in part as described above to the motor nuclei, but pass also in part up the central sensory conducting tracts to the higher ganglia at the base of the brain; where they may cause more complicated reflex acts and pass still higher to the sensory cerebral cortex; where they may give rise to sensations and perceptions (Fig. 35). Here the impulses may apparently stop, or they may be continued from the sensory cortex to the motor cortex and thence a new impulse may pass downward along the pyramidal tract, and thus the involuntary reflex act may be increased or replaced by a voluntary act, or may be abolished (inhibited), voluntarily.

This voluntary abolition of reflex activity (inhibition) may be brought about by a contraction of those muscles which antagonize the muscles taking part in the reflex act, or this latter act may be "inhibited" by a direct action upon the sub-cortical motor cells taking part in it. In addition to this voluntary inhibition, a great variety of nervous activities taking place in almost any part of the nervous system (especially strong, painful impressions), and even the normal process of cerebral activity, will cause a more or less complete inhibition of reflex activity. It seems as though two impulses acting upon a cell at the same time under certain conditions may mutually counteract each other. Therefore, reflex activity is more active in animals in which the brain (or even other parts of the nervous system) is separated from the spinal cord or reflex centers and in human beings when the same result is accomplished by disease (isolation). Curiously enough, when the human spinal cord in its upper part is completely destroyed the reflex activity of the lower parts of the cord is abolished. This phenomenon has not been satisfactorily explained and is in marked contrast to the exaggerated reflexes found in incompletely destructive lesion of the upper portion of the cord.

Conduction of reflex or other impulses along the peripheral nerves is equally rapid whatever may be the intensity, or quality, of the irritation, but conduction through the gray matter is much slower and varies greatly with the intensity and quality of the irritation. The gray matter also possesses the power of summation; so that inactive excitations may become active ones by repetition. The gray matter immediately following its activity shows a "refractory period" of longer or shorter time during which it is inexcitable or exhausted. This indicates that the gray matter accumulates energy during rest, which it discharges when in activity. This refractory period may play its part in rhythmical action. Most reflex acts are purposeful and healthful in their nature. Many of them are absolutely essential for life. They may be divided into the offensive and the defensive.

A destructive lesion of any portion of the reflex arc causes abolition of the reflex acts, as does also a strong irritation of the higher nervous centers. Slight irritative lesions, such as slight inflammations, will cause an exaggeration of the reflex act, as will also and more commonly a lesion which interferes with conduction of nerve impulses (inhibitory impulses) through the central motor (or cortico-spinal) neurons. An irritation, especially a continuous one, even if not very intense, will often cause a tonic spasm or contracture.

VOLUNTARY ACTION, ASSOCIATION REFLEXES (CHART IV)

The anterior portion of the cerebral cortex, which contains innervation (kinesthetic) memories, or motor centers, is connected through bundles of association fibers with the posterior portion of the cortex, in which are memories obtained from the organs of sense. Activity never normally originates directly, or spontaneously, in the motor cortex, but comes to it from the sensory cortex. When a very strong excitation arises in this sensory cortex, as for instance perceptions which are associated with the idea of imminent danger of death, this excitation passes to the motor cortex and thence down through the internal capsule and pyramidal tract and causes movements of flight and self-preservation. This act is as inevitable and as machine-like as is the simplest reflex act. A good swimmer bent on suicide cannot drown himself unless he is weighted or the action of his knees or legs is restricted. Escape from imminent death is for most men an imperative voluntary act. If, however, the danger is less great, as on a battlefield, the excitation leading to flight may be still there, but it may be inhibited by excitation from other associations, such as the idea of shame, love of country, etc., and the two excitations may neutralize each other. It is a question which idea: the fear of death or the love of country and honor, is based on stronger perceptions and wider and stronger associations. Whichever is the stronger prevails.

Ordinarily, when a perception, or memory, suggesting action has sufficient intensity to enter consciousness, the excitation is sufficiently strong to pass along the association fibers and awaken into activity the corresponding innervation memories and, if no other counteracting excitation comes to this latter portion of the cortex, the irritation passes through the great

motor cells in the anterior central convolution and down through the internal capsule and pyramidal tract and the action takes place. Actions resulting from memories are usually weaker than those resulting from the original excitation or perception. When a number of more or less conflicting memories and ideas are in consciousness, some for and some against the action, impulses will be constantly coming to the motor cortex to be either immediately inhibited, or strengthened. The play of the different perceptions, memories and ideas: *the play of motives*, may continue a long time as the person *deliberates* and exercises his *free-will*. This merely means that the different memories, together with the ideas derived from the mass of associated memories which constitute our ethics and those which constitute our emotions, have sufficient intensity to act upon the motor cortex, some as excitants, some as inhibitors. Fresh, allied memories constantly enter consciousness, because of their association with those already in it, and take part in this phenomenon. It is like a debating society in which arguments for and against are presented almost simultaneously, and the stronger argument rather than the will of the judge is the decisive factor. It is probable also that sub-conscious activities may play some part in this process. Eventually the stronger excitation will prevail and the act will be either done or left undone.

A voluntary act, depending upon, and being the result of, the association of ideas, may be described as an *association reflex*. The idea of the apparent freedom of will depends upon the absence of external compulsion and also upon the fact that the action takes place, or does not take place, in accordance with the relative strength of our ideas and desires. The more perceptions and ideas a person has in his memory, the more learned and intelligent he is, the greater, wider and more protracted will be this "play of motives" and the more difficult will be the choice of the resulting action: the association reflex, the victory of any one set of motives. The very learned man is not the man of action. In a child or in an ignorant man, with fewer elements of a choice, the association reflex may be more prompt. When a decision under the same or similar conditions is made a second time, and especially when frequently repeated, the association reflex takes place more and more promptly. In the frequent repetition of acts: practice, the same association conducting channels are being constantly traversed and consequently become better conductors, and the acts become easier to perform: become more or less automatic. It becomes a habit. A large part of our voluntary acts are habitual. It is to be remembered also that the internal sensations, which dominate our "moods," exercise a strong influence over voluntary movements which, under changing moods and altered internal sensations may be very different at different times, although the causal external sensation is the same.

The gray matter: the point of union of the motor and sensory neurons, is in small compass in the sub-cortical centers and hence is well fitted for direct transference for reflex and automatic actions. In the cortical centers the gray matter is spread over a large surface and permits separate, local action, and consequently permits a large number of different memories and ideas, some positive and some negative, to act simultaneously upon the motor cortex and thus either cause or prevent a voluntary action. Both voluntary and reflex acts are for the benefit of the individual. The reflex acts depend upon heredity and evolution. They are the result of the experiences of the individual's ancestors, of the experience of the race (phylogenetic). Voluntary acts depend upon the individual's personal experience (ontogenetic). The difference between the two depends mainly on differences in anatomical structure. In conditions which are new and in which no experience can guide him, an individual's voluntary acts are quite as likely to be detrimental as salutary. His reflex acts almost without exception are salutary.

The innervation memories stored in the motor cerebral cortex are originally acquired from reflex acts. The first voluntary acts of the child (sucking, opening and closing eyes, closing of hand, etc.) are adopted reflex acts, either unmodified or but slightly modified. A young infant does not will to suck milk from his mother's breast. It is a reflex act. But after the infant has experienced the result of this act a sufficient number of times, the sight of his mother, or hunger, may awaken his desire and he will suck the breast voluntarily in consequence of this

active memory. Voluntary motions, or association reflexes, occur early and develop rapidly in infantile life, but occur much earlier, though they develop much slower, in young animals. Chickens run almost as soon as they are born to their mother when she "clucks" for them. The lower animals thus possess at birth, by heredity, a more perfect nervous system; while human infants possess at birth by heredity, one less perfect, but capable of a wonderful development, which results in greater part from personal experiences.

A normal voluntary motion depends not only upon a fairly healthy cerebral cortex (which implies a certain degree of intelligence) and a normal muscle, but also upon the integrity of the two motor neurons: the central (*cortico-spinal*) and peripheral (*spino-neural*), or the upper and lower (461-2). The impulse causing the voluntary contraction starts (as regards its purely motor function) in the cell body of the central motor neuron lying in the anterior central convolution (Fig. 15). It passes along the axon of the cell, which becomes the axis cylinder of a nerve fiber, through the corona radiata, the anterior portion of the posterior limb of the internal capsule (Fig. 17), and emerges from the cerebral hemisphere at the base of the brain in the pes cerebri. It is then covered by the transverse fibers of the pons Varolii, from the lower edge of which it emerges to help form the anterior pyramids of the medulla oblongata (Figs. 20-22). At the junction of the medulla with the spinal cord, these fibers of the anterior pyramids in great part decussate; the completeness of this decussation varying somewhat in different individuals (Fig. 23). Usually the great majority of the fibers decussate and run down through the spinal cord in the posterior part of the lateral column (crossed pyramidal tract), a small minority of the fibers running down in the anterior column of the same side as the pyramid and close to the anterior median fissure (direct pyramidal tract) (Fig. 26). In certain extremely rare cases no decussation takes place. The fibers from both the direct and the crossed pyramidal tracts pass to the groups of motor nerve cells lying in the anterior horns (Fig. 26), and to these cells they communicate their impulses. (The course of these central motor neurons is shown in Fig. 34.) From the group of nerve cells in the anterior horns of the spinal cord, these impulses, thus communicated, pass out along the axons of the cells, which axons become the axis cylinders of the anterior nerve roots, and thus pass along the peripheral motor nerve fibers to the group of muscles innervated by this group of nerve cells in the anterior horn. These impulses enter the muscles by the motor end plates and cause a muscular contraction.

Although the cortical motor centers represent almost exclusively muscles lying on the opposite side of the body, it appears from clinical observation and physiological experiment that the muscles of the body have a bilateral cortical representation. By electrical stimulation of the cortex the muscles on the same side of the body may be made to contract, although a much stronger irritation of the center is needed than is necessary to cause a contraction of the corresponding muscles on the opposite side of the body. Those muscles on both sides of the body which usually act together have especially well marked bilateral representation; so that these muscles are rarely completely and permanently paralysed in unilateral cerebral lesions. A cortical paralysis may affect motion only and may be very circumscribed; two or three fingers, or the thumb only. The actions which are especially lost in the cortical lesions are the purposeful actions which have been slowly acquired as the result of experience and training: actions which are peculiarly voluntary and skilful.

A destructive lesion of either of these types of motor neurons will cause a motor paralysis. If the peripheral motor neurons are destroyed there will be a paralysis both of voluntary and of reflex acts: a flaccid paralysis (252), while if the central motor neurons are destroyed there will result a paralysis of voluntary acts only; the reflex acts persisting and being even increased: a spastic paralysis (253). (For explanation of the increase of reflex activity just mentioned see page 22.)

THE WILL AND WILL POWER

The term "voluntary" motion implies "volition" or "will": some attribute of the individual or personality which controls or regulates the cerebral activities. The will and the freedom of the will stand, however, in direct contradiction both to the law of preservation of force or energy

and to the law of causality. Moreover, we have just learned on page 23 that voluntary motion results from the play of motives and that the strongest sensory cortical activity prevails and causes the resulting motion which seems to be due to our will power only because the perception or memory in consciousness resulting from this "strongest sensory cortical activity" is, in virtue of its strength, in accordance with our so-called will or desire. It seems, therefore, better to call these actions which result from the strongest sensory cortical activity "association reflexes" rather than voluntary acts. As the result of experience in life we acquire desires (see page 11) and each desire can only be satisfied by action. The presence in consciousness of a desire to obtain a certain end or result; which desire is itself the result of cortical activity will by this intense cortical activity excite, influence and usually control those cortical activities, which produce action, thought, study, etc. It is the cortical activity causing the desire which does this, not some external entity: the will.

The act of concentration or attention seems also to call for a will power, but we have learned on page 14 that concentration depends primarily on a strong cortical activity, which the so-called will power and our desires are often incapable of controlling. What has been called the will is probably the dominant cortical activity present in consciousness at any given moment. It depends upon the personality which rests upon many experiences and upon many crystallized experiences in the form of judgments, habits, prejudices, etc., which give our actions stability and consistency; and this sum of consciousness, or even one strong idea in consciousness, the result of a strong cortical activity, may modify the association of ideas and may control both them and the resulting action.

Nevertheless, both in voluntary actions and in efforts of attention, the individual, or the personality, does not seem to himself to be a mere passive spectator, but feels that, although he may be somewhat bound down by experience and habit and prejudice, yet he does exercise a very real and decisive influence upon both these processes, and at least modifies if he does not actually control them. Everyone feels conscious of this power, and it is not altogether satisfactory to dismiss this feeling as a delusion existing in the mind of every member of the human race. It is indeed quite possible that the personality, which is a force produced by chemical process taking place in the sensory cerebral cortex: the result of cellular activity, may in the motor cortex be transmuted back into cellular activity, and thus may promote or inhibit motor action. Just as an electric light, produced by chemical activity within a battery, may be transmuted again upon a photographic plate into chemical action; or as the light of the sun acting upon the chlorophyll in green leaves may break up the molecule of carbon dioxide into its constituent parts: carbon and oxygen.

Personality is a force, and it is hard to conceive of an immaterial spirit as anything else than a force. The primary and essential element in the process still remains the activity of the neuron, and we have already seen that the force generated by one set of neurons may in turn generate, or modify, the activity of another set of neurons. Thus, the personality, which is gradually built up during the life of an individual, may play an active, not a passive, role in the phenomena constituting his conscious life and activity. This statement of what constitutes the will and will-power is in harmony with the subjective feelings of mankind; and yet it is but a restatement in other words of what has been said in the preceding paragraph that "the will is the dominant cortical activity present in consciousness at any given moment," because the personality is also the expression of the sum of the cortical activities present at any given moment.

SPASMS AND CONVULSIONS (CHART IV)

Spasms and convulsions consist in involuntary muscular contraction. They depend mainly upon irritation of the central gray matter, especially the cerebral cortex, and partly upon peripheral irritation.

Passive contracture and Thomsen's disease alone are purely of peripheral (muscular) origin. Many of the tonic spasms are reflex, some are the result of nerve root irritation (meningitis, tumors, etc.) and many are associated with degeneration of the pyramidal tracts.

The result of pathological and experimental investigation makes it evident that epileptic and epileptiform convulsions originate from irritation of the motor cortex. When a slight, but lasting, local irritation of the motor cortex occurs, there results a local spasm, clonic and tonic, which extends from one extremity to another and finally becomes a general convulsion, accompanied in some cases by coma. When the irritation is stronger and especially when it affects both hemispheres there results first a tonic followed by a clonic convulsion and coma. Irritation of other parts of the cortex can also produce epileptic convulsions, if the irritation be strong enough and the motor cortex be intact. Tonic spasms, without clonic ones, may be obtained by irritation of many parts of the central nervous system. The epileptiform convulsion caused by cortical irritation may be accompanied by alterations in the cardiac action in the respiration and in the activity of other internal organs, as in cases of ordinary epilepsy. Indeed, these changes in the cardiac action and in the circulation through the brain may be more essential factors than is the cortical irritation in the actual production of an epileptic attack.

The contractures which accompany cerebral paralyses are due to contraction of the stronger muscles, partly in efforts for voluntary movements from the brain, but mainly reflexly from the spinal cord.

The pathogenesis of many spasms and the localization of their origin, especially of the irregular spasm, are given in the chart.

The various forms of spasms are at times quite difficult to recognize. It requires much experience to be able always to differentiate clonus, tics, athetoid and choreic spasms from one another and from the perversions of motion: tremor, ataxia and apraxia. This is unfortunate because the diagnosis by these diagnostic charts requires that the symptoms be correctly observed and named. The student should compare carefully what he sees with the definitions in the book and should observe as many cases as possible.

APRAXIA, ATAXIA AND TREMOR (CHART IVc)

When an impulse from a cortical motor center passes down to a group of nerve cells in the anterior horns of the spinal cord, it causes a definite synergic contraction of a number of muscles to produce the movement over which this group of nerve cells presides. As soon as this movement commences, a number of sensory impulses pass from the muscles and joints involved to the co-ordinating centers, especially to the cerebellum, and the movement is consequently co-ordinated and orderly. This co-ordination of movements is not inborn. It is acquired by experience and practice. The movements of a new born baby are always ataxic. When the function of the cortical center is impaired there results a paralysis or an *apraxia* (loss of skill) according to the degree of the impairment and when the co-ordinating apparatus is functionally impaired there results ataxia. In either case awkward, ill-adapted and uncertain movements result. There is *asynergy* the muscles taking part in the movement do not act together at the proper time and with the proper relative force to produce an orderly movement.

The motor apparatus, together with its sensory regulation, may be called the executive apparatus and it may be disordered in various ways.

1st. If the motor portion of this apparatus be injured there results a *paralysis* or *paresis*. See Chart IVa.

2nd. If the sensory or regulating apparatus be injured there results *ataxia*. See Chart IVc.

3rd. If what has been learned has been lost or impaired there results *apraxia* or *dyspraxia*. See Chart IVc.

Ataxia: inco-ordination of movement, always depends upon some disturbance of the sensory or regulating apparatus. It occurs in several distinct varieties, depending upon the portion of the sensory nervous system affected.

1st. Peripheral, or dynamic, due to lesion of the peripheral sensory neurons.

2nd. Cerebellar, or static, due to lesion of the cerebellum or its tracts, including the termination of the auditory nerve in the semi-circular canals.

3rd. Cerebral ataxia, to a lesion of the cerebral hemispheres.

1. *Peripheral, or dynamic, ataxia* (280, 644) is caused by an impairment or loss of the complicated sensations conveyed by sensory fibers from the muscles, joints and other tissues which is known by the name of muscle-joint sense (42 and 352). It affects all movements of the parts involved. It is associated with hypotonia (240), which allows an abnormal excursion in passive movements without the resistance normally offered under sudden stretching, and which may allow of abnormal positions of the extremities. The loss of the muscle-joint sense can to some extent be replaced by the sense of sight, which allows the patient to guide his movements by his eyes.

Cerebellar, or static, ataxia (281, 642) is caused by impairment of the function of the great co-ordinating organ: the cerebellum. It affects mainly, or only, walking and standing, which acts resemble those of a drunken man, or become absolutely impossible. The sense of sight gives very little aid in such cases. Movements of the extremities while the patient is recumbent are fairly normal. With cerebellar ataxia is usually associated vertigo; although this latter symptom may not be pronounced.

Cerebral ataxia is due to a lesion of the sensory tracts and centers within the brain. If this lesion involves the sensory tracts in the medulla or pons or crura cerebri, the cerebellar tracts may also be involved and the ataxia may be either cerebellar or dynamic or both. In cases of cerebral hemianesthesia where the lesion is either in the optic thalamus, the internal capsule or the parietal cortex, the ataxia which invariably results is of the dynamic variety and is associated with hypotonia. Such cases of ataxia may be slight in degree and may show great and relatively rapid improvement. When the lesion is in the parietal cortex, the centers for cutaneous and muscular sensibility, ataxia results, because of the loss of those sensations which are essential for the proper guidance of voluntary movements. In tumors of the frontal lobe, whether cortical or sub-cortical, ataxia is a common symptom and is of the cerebellar type; being doubtless due to involvement of the fronto-cerebellar tract. In cerebral ataxia it is evident that the patient is trying to execute the movements and knows what he wants to do, but he executes them awkwardly.

Apraxia (282) may result from the loss of the purposeful idea which should prompt a given action. In lesions of the posterior central convolution or of the supra-marginal gyrus this idea cannot be formed (sensorial apraxia or agnosia), in which case the action which should follow the idea cannot originate; or when this idea is formed the memory is quickly lost (amnesic apraxia), in which case the appropriate action is begun, but never completed. In lesions of the anterior central convolution, or of the area immediately anterior to it, the purposeful idea may be present, but the innervation memories necessary for the production of the appropriate action are lost; so that the action cannot be performed (motor apraxia). When the association fibers connecting the anterior and posterior central convolutions are the seat of lesions, the appropriate action will not occur, or a somewhat similar action may be substituted for it (associative apraxia).

In any organized society much results from imitation and from instruction. Certain complexes of innervation feelings become by practice so firmly united, that what was at first done with difficulty and imperfectly, becomes easily and perfectly done. These innervation complexes are not inborn (although their anatomical sub-structure may well be), but are learned, are acquired by practice. These innervation complexes become memories (kinesthetic memories). Innervation memories may be conscious in early life when first learned, but may be unconscious later. Many of them may never enter consciousness. As long as these memories persist the corresponding action may be performed, consciously or unconsciously, as the final result of sensory impulses exciting them. Many complicated acts are not performed often enough to form an innervation complex, but must be performed consciously and with constant sensory guidance from many parts of the brain simultaneously (sight, muscle sense, touch, etc.).

Tremor (250) may be caused by rapid rhythmical interruptions of the innervation impulses passing to the muscles or by a failure of a proper proportion or equilibrium in the innervation of the muscles and their antagonists. Tremor usually ceases during sleep and is usually increased

by mental excitement; although a very powerful emotion may arrest the tremor temporarily. It seems to be always of central origin. Clonic spasm from exaggerated reflexes must not be confounded with a coarse tremor.

SPEECH (CHARTS IVc AND XIII)

The most complicated and important of all voluntary acts is speech. Speech and the allied functions: reading and writing, are peculiar to human beings and are the result of much instruction in the line of imitation and study. These functions, therefore, depend upon a healthy brain. If a child has such an imperfectly formed brain that he is an idiot (743), he consequently cannot speak. The perfection and content of speech, reading and writing depend upon education; being more imperfect the less the education and training and are, therefore, often quite abnormal, or even absent, in the defective and feeble-minded (750, 1088, 1092) and in hysteria (747-8) and insanity, especially in adolescent insanity (1096), in dementia (1103), in coma (745) and in insanity with diffuse cortical changes in the speech area (1104).

The power of speech is of enormous importance in the development of the race. It is the one factor which has enabled the human race to so far outstrip all other animals that it seems to form an entirely different order of beings from them. The spoken, and still more the written, word allows man to make his own all the experience, knowledge and wisdom of his ancestors and contemporaries, and raises him, thus, far above his own limited individual experiences.

Like all knowledge, the art of speaking, reading and writing is acquired from sensory impressions. The art of speech is derived from the sense of hearing; so that when a child is born deaf, or acquires deafness in the first two or three years of life, he is also dumb: a deaf mute (744). A deaf mute can be taught to speak only very imperfectly, and then only by the sense of sight, or much more rarely by touch (Helen Keller). The art of reading and writing is derived partly from the sense of hearing and mainly from the sense of sight. If a child is born blind, or acquires blindness in the first few years of life, he can learn to read only books printed in a peculiar way, and then only by the sense of touch and hearing; the sense of touch replacing the sense of sight in these cases.

It is evident, then, that the perceptions and memories of spoken words are of fundamental importance in the art of speaking. These perceptions take place and these memories are stored, in right handed persons, in the posterior half of the left superior temporal convolution and in the posterior portion of the left island of Reil (Fig. 15); so that lesions of this area cause a profound disorder of speech: sensory aphasia (772). From this portion of the cortex impulses pass along association fibers (the fasciculus uncinatus) lying in the external capsule to the base of the left inferior frontal convolution (Fig. 15) and to the anterior portion of the island of Reil. A lesion in this region also causes a profound disorder of speech: motor aphasia (771).

The distinction between motor and sensory aphasia is not always easily drawn. In some cases when a patient is unable to speak a desired word it may be very difficult to decide whether he has forgotten the innervation memories necessary to speak the word (motor aphasia—221), or has forgotten the word itself (sensory aphasia—222). In the latter case he may be able to repeat the word when he hears it spoken. Lesions of the external capsule, in which run the association fibers connecting the centers of sensory and motor speech (the fasciculus uncinatus), also cause a profound disorder of speech (conduction aphasia).

Perceptions of written or printed words are formed and their memories are stored, in right handed persons, in the cortex of the left occipital lobe, and from this area impulses pass along the association fibers lying beneath the angular gyrus to the base of the left inferior frontal convolution and the base of the left middle frontal convolution, where are stored the innervation memories of speech and writing respectively. Therefore, deep lesions in the region of the left angular gyrus in right handed persons will cause a complete alexia (773) and an incomplete agraphia (776). The area of the cortex in the left hemisphere described above, including the bases of the middle and the inferior frontal convolution, the island of Reil, the posterior half

of the superior temporal convolution and the angular gyrus is called "the zone of language" and is the cortical center, or psychic center, for the faculty of language.

In addition to its cortical center, speech depends upon the integrity of the muscles and nerves which move the lips, tongue, soft palate, larynx and those concerned in respiration. In lesions of these muscles and nerves and of their nuclei in the medulla and pons and of the pyramidal tract, speech may be abolished (anarthria) or pronunciation impaired (dysarthria), whether in consequence of paralysis or of inco-ordination, or of spasm (as in stuttering). Reading and writing may be similarly abolished or impaired in lesions of the peripheral nerves or of their nuclei in the optic thalamus or in the anterior horns of the cervical enlargement of the cord or of the fasciculus of Gratiolet or of the pyramidal tract.

Dysarthria might also be due to a cortical paralysis of the pneumogastric nerve, but the laryngeal muscles have a bilateral cortical representation; so that if one cortical area be injured the corresponding area of the other hemispheres can carry on the function of speech perfectly. There is, therefore, no laryngeal paralysis, or consequent dysarthria, due to any lesion within the cerebral hemispheres, unless the lesion be very extensive and involves both hemispheres (pseudo-bulbar paralysis).

AUTOMATIC MOVEMENTS (CHARTS III AND XVI)

This term is applied to two quite different sorts of actions. In one sense automatic, or autochthonous, acts are reflex acts which originate, not from external, but from internal, or organic, excitations or irritations. One of the best examples of this activity is the respiratory act. Such acts are very numerous and carry on the nutritive activities of the body.

The name is also applied to voluntary acts which have been learned with more or less difficulty, but which have been enacted so often that they can be performed without consciousness. Such acts are walking, writing, piano-playing, smoking and many others which can be very perfectly performed unconsciously, although each one can also be enacted consciously and usually is so done.

TROPHIC INFLUENCES (CHART XVII)

The nervous system exercises an important trophic influence over many of the tissues of the body, in addition to influences over their blood supply through the vaso-motor system. This trophic influence can be divided into two great divisions, motor and sensory. When the motor nerve cells of the central or peripheral motor neurons are degenerated or destroyed (as in lesions of the nerve fibers or of the motor cells, of which these nerve fibers are the axons), the nerve fibers springing from such degenerated cells undergo a rapid degeneration, as do also the muscles, in which these nerve fibers terminate; and in early life when there is motor paralysis, or immobility of parts of the body from any cause, these parts fail to grow normally.

When the sensory nerves are degenerated, as in syringomyelia, myelitis, tabes, lesions of the spinal ganglia or of the ganglia at the base of the brain, etc., in consequence of the anesthesia, the body is no longer protected, by reflex and voluntary acts, from the many traumatisms to which it is frequently subjected and therefore ulcerations, arthropathies, ulcerations of the cornea and other trophic lesions result.

Some of the ductless glands, especially the pituitary and the thyroid, when hypertrophied or atrophied as regards their glandular structure, also produce widespread trophic disorders.

THE CEREBRO-SPINAL FLUID (CHARTS VIII AND XIX)

The central nervous organs (brain and spinal cord) are bathed in a fluid called the cerebro-spinal fluid. This fluid is secreted or transudes from the choroid plexus within the ventricles of the brain and thus may contain substances which are in the blood. It passes out of the ventricles at the inferior angle of the fourth ventricle, through the foramen of Magendie. If from any cause (tumor, meningitis, etc.) the foramen of Magendie is occluded, this fluid, constantly secreted, cannot escape from the ventricles and dilates these cavities more or less ac-

ricing as the sutures of the skull are ossified less or more completely; thus producing internal hydrocephalus. The cerebro-spinal fluid passing out of the foramen of Magendie becomes the sub-arachnoid fluid, which lies in the meshes of the tissue forming the deeper layers of the arachnoid. In this situation it can receive products of any inflammation of the meninges: albuminous substances (globulin) and cellular structures (leucocytes in acute, and lymphocytes in chronic, inflammations); so much so as to be cloudy or even purulent. The specific germs of the various forms of meningitis can often also be detected, as well as blood in hemorrhage and pus in abscess. In tertiary and quaternary syphilitic meningitis the Wasserman reaction is usually positive.

The cerebro-spinal fluid is obtained by lumbar puncture and the rapidity of its escape is evidence of the tension which it is under, which tension can more accurately be measured by a manometer. When the cerebro-spinal fluid is increased in amount, as in meningitis, or when a foreign body, as a tumor, is within the cranial or spinal cavity the tension of the fluid is usually increased. The examination of this fluid is, therefore, of much importance in disease of the cerebral and spinal meninges and in other intra-cranial and intra-spinal conditions.

ELECTRICITY AND THE NERVOUS SYSTEM

Nervous conduction, although it has some analogies with electrical conduction, is due to an entirely different form of energy. But when nervous action takes place, whether in a peripheral nerve or in a central ganglion, there always occurs an electrical current through the nerve or ganglion in the opposite direction. So constant and delicate is this reaction, that it has been used to prove the presence of nervous activity. Moreover the electric current, both Galvanic and Faradic, can be conducted along nerve fibers, and changes in the tension of electricity so conducted in the nerve fibers cause contraction of the muscles in which they terminate; as is shown in Chart VII. The muscle fibers also respond directly to changes in intensity of a galvanic current, but not to those of a Faradic current.

All forms of electrical energy are excitants for all the sensory organs, acting not so much upon the end-organs as upon the nerves themselves.

Other forms of electricity, especially static electricity and high frequency currents, are used as therapeutic measures but have no diagnostic value.

CHART I
Case-Taking

METHODS OF EXAMINATION OF PATIENTS SUFFERING FROM NERVOUS DISEASES

Errors in diagnosis result more frequently from imperfect observation than from faulty reasoning.

	Data derived from	
	QUESTIONING.....	see chart I a.
	INSPECTION.....	see chart I b.
	PALPATION.....	} see chart I c.
	PERCUSSION.....	
Methods of Examining and Testing Patients.....	ELECTRICITY.....	} see chart I d.
	LUMBAR AND BRAIN PUNCTURE.	
	OPHTHALMOSCOPY.....	
	LARYNGOSCOPY.....	
	THERMOMETRY.....	

CHART I a

Questioning

Comprising Numbers 1 to 18

(Note)—The examination of every patient, who is conscious and intelligent, begins with a history of his health and of that of his ancestors. This is an important source of information, although usually less so than are the results of the physical examination. The taking of a reliable clinical history is something of an art, but at best we are absolutely dependent upon the truthfulness of the patient, as we rarely have means to check his statements by information from other sources. It is important to put the patient at his ease and to gain his confidence. The patient is vitally interested in his own case and it is best to let him tell his own story of his illness in his own way, without interruption; then to question him further about his illness, more especially and fully in regard to the organ probably affected, but also concerning the function of the other organs of the body. This done, he should be questioned as to his previous illnesses, occupations, etc., and finally as to any special prevailing illness in his ancestors or relatives. It is important to ask as few leading questions as possible. Questions in regard to personal habits and venereal diseases should only be asked when absolutely alone with the patient, and then in a manner which assumes that all men are guilty of indiscretions. During our taking of the clinical history we should have the patient under close observation and can thus form a good judgment as to his manner and general mental and physical characteristics.

METHODS OF EXAMINATION OF PATIENTS SUFFERING FROM NERVOUS DISEASES

QUESTIONING

METHODS OF TESTING

- 1 Allow the patient to tell the story of the illness without interruption. Then
History of present illness. ascertain the exact date and manner of onset (sudden or slow, prodromata, etc.) and the exact sequence of symptoms. Inquire into all details which may concern the case (headache, pain, paresthesiae, vertigo, insomnia, mental condition, emotions, memory, special senses, paralyses, spasms, fits, disturbances of organic reflexes, loss of weight and strength, etc.), whether of recent or of old date. Seek for any possible cause (injury, poisons, drugs, infections, worry, mental or physical overstrain, shock, etc.). Be careful not to suggest answers to nervous people. Inquire into previous treatment and its effect.
(Chart II)
- 2 Ascertain the occurrence, in the present, or a past, generation of the family, of
Family and personal any nervous diseases, especially the neuroses (neuralgia, epilepsy, hysteria, insanity, suicide, drunkenness, etc.), or of syphilis, or tuberculosis. Inquire as to consanguineous marriages. Note patient's age, full address, race, his mental and physical development in school life, occupation, habits (alcohol, drugs, venery, masturbation, etc.), dwelling and previous illnesses, such as rickets, infectious diseases, chorea, fits, tuberculosis, syphilis (use discretion in this inquiry: ask about sore throat, skin rashes, miscarriages, etc.), and injuries at birth or later. Ascertain the condition of other organs (cancer and tuberculosis).
history.
(Chart II)
- 3 Patient may lie in a stupor and make little or no response to questions, noises, shaking, pin pricks, or strong sensory irritations of any kind. He may appreciate neither his surroundings, nor his acts, nor the time and place, nor his own individuality. He can remember, after recovery, nothing of what happened while he was unconscious. There are all possible grades in impairment of consciousness from complete coma to a slight lack of attention and an inability to collect his thoughts. This can be learned by conversation with him.
Consciousness.
(Charts III & XVI)
- 4 Patient's conversation and manner may show that his brain acts in an abnormal way and that he entertains abnormal perceptions and ideas (hallucinations, delusions, compulsory acts and ideas, etc.). Ascertain if a change has taken place in the patient's normal mental state, and when. Note whether patient is elated, active, loquacious; or dull, inattentive, sluggish, distracted, evasive, suspicious, and why. Some cases may require prolonged observation. At times irritating questions may be desirable, in order to excite the patient.
Sanity.
(Charts III & XVI)
- 5 In testing a patient's intelligence, we test his *general knowledge* by asking him to name the different days and the different months and by arithmetical, geographical, political and historical questions. His *power of observation* by showing him a number of things and asking him later to describe them. His *power of attention* by asking him to add a long column of figures or underscore a letter wherever it occurs in a page of print. His *power of comprehension* by asking him to explain something he has read or heard. His *association of ideas* by giving him a word and asking what other ideas it suggests to him. His *mental reaction time* by the time he takes to solve problems, or to name an object, the picture of which is shown to him. His *moral sense* by questions in ethics.
Intelligence.
(Charts III, XIII & XVI)
- 6 An apparent defect in intelligence may be due to lack of attention, or may be shown by further questioning, by having him repeat long phrases, execute verbal and written commands and name objects shown to him, to be due wholly or in part to a loss of memory; either general (amnesia), or local (aphasia), especially to a failure to understand what is said to him (sensory aphasia); while reason and judgment are normal. Test memory for remote, as well as for recent, occurrences. Test his memory of statements made a few minutes previously, or of events of the day before, or of years before.
Memory and understanding.
(Charts III, XIII & XVI)
- 7 Patients may show by their conversation, if suitably guided, or by their manner, or by both, whether they are emotional or not. The emotional state of the patient and the mental characteristics discussed just above, can often best be learned from the statements of friends and relatives. Curious fears, the so-called "phobias," (235) are often present.
Emotions.
(Charts III & XVI)

QUESTIONING (Continued)

- 8
Speech.
(Charts III,
XIII & XVI) Patient's speech may be entirely absent (anarthria) or altered and very defective, i.e., rational or irrational; there may be limited vocabulary or use of wrong word (aphasia), poor articulation (dysarthria), tremor in voice, monotonous, scanning speech, omissions of syllables and words. Speech is tested by interrogation and spontaneous (voluntary) speech. Test also patient's understanding of letters, words and phrases spoken to him, his executing spoken and written commands, his picking out objects named; and have patient name objects, give sequences, i.e., numbers, days of week, months, etc., and repeat catch phrases, as "Round the rough and rugged rock the ragged rascal ran," etc.

- 9
Reading.
(Charts III,
XIII & XVI) Ask the patient to read aloud, even short sentences, words, or letters only. Note any defect either in utterance or understanding.

- 10
Writing.
(Charts III,
XIII & XVI) Ask the patient to write, spontaneously, from dictation and from copy. Have him write the names of objects shown him. Note any defect in the character of the writing or in the ideas expressed.

- 11
Stereognosis.
(Charts III,
VI & XXII) Ask the patient to name objects placed in his hand, his eyes being shut, after excluding anesthesia. Even without feeling them all over and without moving them about in his fingers, a normal person should be able to recognize many objects (metals, cloth, etc.) merely laid against the skin of his hand, face, foot, lips, etc. Stereognosis may, therefore, in exceptional conditions, be tested, although less perfectly, in other parts than the hands.

- 12
Sight.
(Charts VI &
XIV) Ask the patient to read small print or Jaeger's test type at reading distance (10 to 16 inches, according to age, refractive conditions, etc.) and Snellen's test letters at twenty feet. If patient cannot read the appropriate line at twenty feet the loss of vision is expressed by the number of feet from the chart at which he can read this line divided by twenty. Thus at ten feet the vision would be expressed by $\frac{1}{10}$. In great defect of vision the patient may be able to see only dimly the hand moved before his eye, or may only be able to distinguish between light and darkness.

- 13
Color sense.
(Chart VI) Ask the patient to match different colored worsteds.

- 14
Field of vision
for white
and colors.
(Hemianopia)
(Charts VI
& XIV) Place the patient with back to the window or light and have him close his left eye and with his right gaze at the observer's left eye. Then let the observer move his hands about in a plane mid-way between himself and the patient; so that each should see the hand at the same instant as it comes into the field of vision. The observer can see if the patient's eye wanders from his own and recall it. Test left eye in same way. If any defect in field of vision is suspected, use a perimeter. With a perimeter not only the field of vision, but also, by using different colored papers, the color field can be mapped out. Normally the color field is largest for blue, then for yellow, orange, red, green, etc., in the order named. If this order is changed there is said to be an "inversion of the color fields" (849). Normally the lines limiting the different color fields are everywhere separate from each other. If they touch or cross there is "interlacing of the color fields" or "dyschromatopsia" (849).

- 15
Hearing and
tinnitus
aurium.
(Charts VI &
XIV) The patient's hearing may be tested by voice, watch, or tuning fork. Be sure there is no wax in the ear. Galton's whistle should be used for testing high and low notes. Each ear should be tested separately. Bone conduction is tested by holding watch or tuning fork firmly on skull. Normally a tuning fork, which, held on mastoid ceases to be heard, can still be heard when held close to meatus (Rinne's test). Normally a vibrating tuning fork, held on center of forehead, is heard equally in both ears. If heard best in the deaf ear (positive) the lesion is in external or middle ear. If heard best in the normal ear (negative) the lesion is in inner ear or in auditory nerve (Weber's test). We also ask about ringing in ears (tinnitus aurium).

- 16
Smell.
(Charts VI &
XIV) Ask patient to name from its odor any fragrant substance (such as asafoetida, cloves, peppermint, etc.) held for a moment beneath each nostril in turn, the other being closed. Ammonia and acetic acid should not be used in this test.

- 17
Taste.
(Charts VI &
XIV) Ask the patient to point to the name on a printed card of the taste of a strongly bitter, sweet, salt or sour solution touched from a medicine dropper, or a camel's hair brush, to one side after the other of the protruded tongue. The tongue should be well washed between each test.

- 18
Sleep. The amount of sleep which the patient gets in the twenty-four hours is always an important question. Insomnia (agrypnia) is present in many nervous diseases and is apt to be exaggerated by patients; so that their statements should be controlled, when possible, by those of the nurses or relatives. Many symptoms, especially fears, are worse at night: "Pavor nocturnus of children."

CHART Ib

Inspection (mainly)

Comprising Numbers 20 to 42

METHODS OF EXAMINATION OF PATIENTS SUFFERING FROM NERVOUS DISEASES

INSPECTION

METHODS OF TESTING

- 20
Facial expression and general appearance and behavior.
(Charts XVI & XVII)
The expression of the patient's face indicates, in most cases, the degree of his intelligence and his emotional state (sad or gay or anxious), and also may suggest the presence of certain diseases and conditions; such as myxedema and cretinism (1163-4), acromegaly (1183), scleroderma (1165), exophthalmic goitre (1193), paralysis agitans (677), myasthenia (553), nasal obstruction, atheroma of temporal arteries, notched teeth, hazy cornea, and the saddle-back nose of syphilis, etc. His general appearance and behavior often indicate his power of self restraint (inhibitory power, breeding), or the existence of hallucinations (213) of sight, hearing, touch, or of compulsory acts (218).
- 21
Walk.
(Chart XIII)
The walk of the patient may suggest the presence of hemiplegia (254), paraplegia (257), local paralysis (259), ataxia (motor or cerebellar) (248), spasm (242), atony (252), paralysis agitans (677) and other tremors (250), pseudo-hypertrophic paralysis (500), hysterical paralysis (527), foot drop, (bilateral in multiple neuritis and lead palsy, unilateral in acute anterior poliomyelitis), weakness, exhaustion, etc.
- 22
Skull. †
(Chart XVI)
The skull should be observed as to type (brachy- or dolicho-cephalic, round or long heads), size (microcephalic—small, macrocephalic—large), rickets (box shaped), general or local hydrocephalus (bulging—posterior or anterior), fontanelles and sutures, asymmetry, tumors, etc.
- 23
Vertebral column.
(Chart X)
The spinal column should be observed as to curvature (angular or lateral), scoliosis, kyphosis, spina bifida (occulta), deformity (dislocation), Pott's disease, tumor tenderness (by palpation), etc.
- 24
Eye.
(Charts V, VI & XIV)
Note the existence of arcus senilis, the condition of pupils (unequal, anisocoria (341), myosis (340), mydriasis (339), and irregularity), the presence of keratitis or iritis, prominence of eyeballs, nystagmus, squint, ptosis, paralysis, etc.
- 25
Pupillary reflex to light.
(Charts V & XIV)
Note whether each pupil, the other eye being covered, dilates and contracts as the eye is alternately shaded by the hand and exposed to light, or an electric light is flashed into it; vision being constantly fixed upon some distant object. When a pupil contracts to light (direct reflex) the pupil of the other eye also contracts (consensual reflex).
- 26
Hemiopic reflex.
(Charts V & XIV)
Note whether the pupil contracts as light is flashed on each half of the retina alternately. A ray of light collected by a lens should be used in this test. This reflex is difficult to obtain, and not entirely reliable.
- 27
Pupillary reflex to accommodation.
(Charts V & XIV)
Note whether the pupil dilates when the patient looks at a distant object and contracts when he looks at one so near his face as to require convergence of the eyes. This test can be made on a blind man by having patient first converge his eyes and then make the axes of his eyes parallel, by imagining that he is looking at a near and then at a distant object.
- 28
Double vision, diplopia.
(Charts VI & XIV)
Note which eye deviates, however slightly, from the direct axis of vision and which eye lags more or less on movement of eyeballs in following the moving finger. Place a colored glass before the affected eye, move a bright object (candle) throughout the field of vision and have the patient note the relative position of the two images. The colored image will of course be the one seen by the affected eye.
- 29
Secondary deviation of the sound eye.
(Chart XIV)
Hold a card close in front of the sound eye. Have the patient look at an object so held that the weakened muscle must be brought into action. The sound eye covered by the card will be observed to move too far and when the card is removed the sound eye will quickly move back into proper position.
30.
Nystagmus.
(Charts IV & XII)
The oscillation of the eyeball which constitutes nystagmus is often plainly to be seen. Extreme deviation of the eyeballs in one direction or the other makes it more evident, and at times demonstrates a nystagmus not otherwise apparent. If present, nystagmus is usually recognized while making the two tests 28 and 29. It should not be confounded with the irregular jerky motion of a weakened ocular muscle attempting to move the eyeball.

INSPECTION (Continued)

- 31 Tremor. (Charts IV & XII) Note any tremor of lips, tongue, or other parts of the body. Note its frequency, amplitude, its relation to voluntary movements and whether it is associated with muscular rigidity. In testing for tremor, ask patient to hold arms extended before him or over his head with fingers spread and motionless.
- 32 Convulsion and spasm. (Charts IV, XI & XII) Note any convulsion (269), spasm (245-6), contracture (263-4), athetosis (271), choreiform movement (272), etc., which may be present. These various forms of spasm are often difficult to recognize and differentiate from each other.
- 33 Paralysis (motor). (Charts IV, X & XIII) Note any obvious paralysis, such as ptosis. Note the naso-labial fold and the height of the angle of the mouth on each side. While under close inspection, patient should be requested to execute every possible motion: i.e., wrinkle forehead (look upward, or open eyelids held closed by observer), frown, open and shut each eyelid, move eyeballs up and down and to either side (note whether upper eyelid follows eyeball well downwards), whistle, laugh, distend cheeks, raise upper lip and each angle of mouth, protrude tongue straight and move it in all directions, raise uvula in phonation, close jaws and move chin forwards and jaw laterally, contract strongly all muscles of face at once, move head backwards, forwards and towards each shoulder and shake it, bend body in all directions, raise arms vertically, raise shoulder, adduct and abduct arm, flex and extend elbow, wrist and each finger, spread fingers, adduct, abduct, flex and extend thumb, pronate and supinate forearm while elbow is flexed, stand on each leg, raise body on tiptoes, adduct and abduct thigh, flex and extend thigh, leg, foot and toes.
- 34 Paresis. (Charts IV, X & XIII) Make strong resistance to above mentioned movements while patient is executing them: i.e., pull on eyelids, on one angle of mouth, resist movements of jaw, or of bending head or body, or of flexing, extending, adducting and abducting joints, compare the strength of the paretic muscle with that of a similar healthy one, when possible, with its fellow of the opposite of the body. For future comparison, etc., the strength of the paretic muscle can be registered by dynamometers, of which the most practical is the one for the hand grasp. Or sufficient weights may be placed on hand, foot or head to overcome the attempted movement.
- 35 Myasthenia. (Chart IV) Note whether patient tires easily on repeated or continuous activity of any set of muscles.
- 36 Diadocokinesis. (Chart IV) Note whether patient can alternately extend and flex joints quickly and repeatedly. Test especially rapid alternate supination and pronation.
- 37 Ankylosis. Note whether any joint is rigid, so that it cannot be moved. Ascertain the cause of the rigidity, whether bony union, contracted muscle or contracted scar tissue (muscle, ligament, skin, etc.).
- 38 Contracture. (Charts IV & XI) Note whether any muscle is contracted with consequent impaired motility of the joint and whether this contracture can be overcome by force, with or without etherization (active contracture), or not (passive contracture).
- 39 Muscle tone. (Charts IV & X) Note whether muscles are firm or flabby, and whether or not resistance is offered to rapid passive motions of joints while the patient tries to make no voluntary resistance. Normally there is slight resistance. In disease the resistance may be altogether absent (atonia), or weak (hypotonia), or strong (hypertonia).
- 40 Trophic lesions. (Chart XVII) Note whether any muscle shows atrophy or hypertrophy, or fibrillary contractions, or if there is any arrested development or trophic lesions of other tissues (especially ulcers, herpes, glossy skin, abnormalities of nails, etc.).
- 41 Co-ordination (synergy). (Charts IV & XII) Note whether complicated movements are executed in an orderly manner while the patient's eyes are closed. Ask patient to walk, touch point of nose with finger tip, pick up objects, write, touch knee with heel of other foot, hold foot steady in one position, trace a circle in the air with foot, walk backwards, walk along a line, stand on one foot alone, or on both feet close together, either side by side or one in front of the other (Romberg's symptom), stand on tiptoes or on heels, stand on one foot and trace a circle on the floor with the toe of the other foot. All these tests should be made both with eyes open and shut.
- 42 Muscle and joint sense. Deep sensibility (bathyes-thesia, kinesi-thesia). (Charts VI & XII) Note whether patient, with his eyes shut, can tell whether his joints are flexed or extended, or can duplicate with one extremity the position in which his other is placed. Note whether he can estimate weights correctly or can grade by weight loaded balls correctly. Note whether he can locate his extremities in space. To test this, his eyes being shut, an extremity after being moved about is held in one position and he is told to turn his head and eyeballs so that when he opens his eyes he shall be looking directly at his thumb or great toe. When he opens his eyes it will be plain to see whether they are directed right or not.

CHART I c
Palpation and Percussion
Comprising Numbers 45 to 68

METHODS OF EXAMINATION OF PATIENTS SUFFERING FROM NERVOUS DISEASES

PALPATION AND PERCUSSION

METHODS OF TESTING

- 45
Circulation and respiration.
(Chart XVII) Note the color of the skin, the pulsation of arteries in neck, the condition of the jugular veins and the frequency and regularity of respiration, especially Cheyne-Stokes' respiration (435), whether respiration be costal or abdominal, or diaphragm be immobile, unilaterally or bilaterally.
- 46
Pulse.
(Chart XVII) Note pulse of patient as to frequency, volume, tension (best tested by tonometer or sphygmomanometer) and irregularity in rhythm and force.
- 47
Difficulties in sensory testing.
(Chart VI) The result of all sensory tests (and the same is true in regard to tests for many mental symptoms) depends upon the patient's truthfulness. Deception is always possible and even with the most truthful patients the tests require much time and the results are often contradictory, especially so in excitable and in uneducated patients, who cannot fix their attention continuously. Nothing should be present to distract the patient's attention and his skin should be warm. In some nervous diseases the patient has occasional, spontaneous sensations which interfere with the tests. Most patients under the education of repeated tests become more acutely sensitive. On the other hand, tests too long continued tire the patient and give rise to contradictory results. It is to be remembered that the sensibility of the skin both for tactile and painful impressions varies greatly in different parts of the body and in different individuals.
- 48
Tactile sensibility.
(Charts VI & XIV) With the finger tip (or with a smaller and lighter object, such as the head of a pin, a camel's hair brush, a pledget of cotton, a hair, etc.), touch the patient's skin lightly, having told him to say "yes" every time he feels the slightest touch. Or the patient may describe figures (space sense) traced on his skin with ink (to prevent dispute or doubt). A pledget of cotton is better for accurate testing than is the finger tip or a pin, because with the cotton the pressure sense (49) is eliminated. Of course, during all sensory tests the patient's eyes must be closed or covered. In some cases of hysterical anesthesia, if the patient is told to say "no" when she does not feel the touch, she will say "no" only at the instant she is actually touched within the anesthetic (?) area; showing that sensation is not abolished, although it may well be abnormal. Tactile sensibility, or, more properly, "space sense," or "localizing sense," (53), may also be tested with the esthesiometer; a pair of blunt dividers, by which it is noted how far the points may be separated and yet be felt as one. This distance varies greatly in different parts of the body (at the point of tongue it is one m.m., at finger tips two m.m., along back and on upper part of arm and thigh it is sixty-five m.m. The distance is smaller transversely than longitudinally on the extremities. Neither this compass aesthesiometer, nor Herring's aesthesiometer gives more valuable results than the pin-head tests. When mapping out an anesthetic area commence in the anesthetic area and work towards the normal skin. Do the reverse in mapping out hyperesthesia; i.e., from normal skin to hyperesthetic area. The electro-cutaneous test can be more accurately measured, but is of little practical value.
- 49
Pressure sense.
(Chart VI) Note whether patient can estimate correctly the amount of pressure exercised by the finger pressed against the skin, or by weights laid upon it.
- 50
Painful sensibility.
(Charts VI & XIV) Note whether patient feels pain when pinched, or when skin is pricked by fingernail, pin-point, or other sharp substance. Many instruments have been devised for measuring more or less accurately the intensity of the painful impression.
- 51
Retardation of conduction.
(Chart VI) Note whether the painful sensation is felt immediately upon, or some seconds after, the painful contact.
- 52
Persistence of sensation.
(Chart VI) Note whether the painful sensation persists a longer time, after the painful contact has ceased, than is normal.
- 53
Localization.
(Chart VI) Note whether the point of contact, tactile or painful, can be localized correctly by the patient either by description or by pointing; his eyes, of course, being shut.
- 54
Double sensation and polyesthesia.
(Chart VI) Note whether a single tactile or painful contact causes two (double sensation) or more, sensations (polyesthesia).

PALPATION AND PERCUSSION (Continued)

- 55**
Temperature sense.
(Chart VI) Touch the skin at numerous points alternately with small test tubes, one filled with hot, the other with cold, water, or with hot and cold bodies (spoons) of the same size and form. Certain points of the skin are especially sensitive to heat; others to cold. It is well, therefore, to test for heat and cold separately.
- 56**
Pallesthesia.
(Chart VI) Note whether the patient feels the vibration of a tuning fork (vibration sense) pressed so firmly on the skin that the vibration can be transmitted through the underlying bone (osseous sense).
- 57**
Cutaneous reflexes.
(Chart V) Stroke or scratch, as softly as will suffice, with finger nail or head or point of pin, the skin of the sole of the foot (plantar and Babinski), or a buttock (gluteal), or the inner side of thigh (cremasteric), or the side of abdomen (umbilical), or the hypochondrium (epigastric), or interscapular region (interscapular), or stroke firmly along the postero-internal border of the tibia (Oppenheim's reflex) and note the resulting movement. The muscle itself must be felt and watched in cases where the resulting contraction is too slight to move the part.
- 58**
Mucous membrane reflexes.
(Chart V) Touch with finger, straw, brush, or probe, the cornea or conjunctiva (conjunctival), or mucous membrane of nose (nasal), or palate (uvular), or pharynx (pharyngeal), and note the resulting movement.
- 59**
Vaso-motor reflexes.
(Charts V & XVII) Note the pallor or redness of the skin, also rapid changes and flushings with or without irritation, such as scratching with a pin or fingernail (dermographia).
- 60**
Ankle-clonus.
(Charts V & X) With leg relaxed, semi-flexed and well supported, strike or press the sole of the foot quickly, firmly and continuously upwards and note whether the foot oscillates or not. This clonus occurs at times spontaneously when the toe and not the heel rests on the floor ("spinal epilepsy").
- 61**
Knee-jerk.
(Charts V & X) While patient is sitting on a chair with legs crossed, or better on a table with legs hanging free, or is lying in bed on his back with knees flexed, strike the ligamentum patellae a sharp blow with the finger, edge of hand, book or percussion hammer and note whether the foot flies forward. The amplitude of the excursion of the foot is not alone a safe guide to infer increase of knee-jerk, but rather its vigor, its quickness, and the presence of two or three additional oscillations as the foot falls back again. Even a continuous oscillation, or clonus, occurs in some cases (the so-called "spinal epilepsy"). More common than this clonus is a simultaneous contraction of the adductors of the other thigh when the knee-jerk is exaggerated. In order to obtain this reflex the observer must make sure that the muscles of the legs are completely relaxed. The extensor femoris muscle must be observed and felt in those cases where the resulting contraction is too faint to move the leg. Knee-clonus may be obtained in suitable cases by grasping the patella from above and pulling it sharply downwards.
- 62**
Achilles reflex.
(Charts V & X) While patient is kneeling in a chair with his feet projecting free, the tendo-Achillis should be strongly struck with a percussion hammer and the movement of plantar flexion noted. Where the patient cannot kneel the leg may be supported in any position which relaxes it and the tendo-Achillis struck.
- 63**
Dorsal foot reflex.
(Chart V) When the dorsum of the foot is struck sharply over the 4th or 5th metatarsal bones, note the dorsal (normal) or plantar (pathological) flexion of toes (Mendel-Bechterew's reflex—320).
- 64**
Elbow and wrist reflexes.
(Chart V) The arm being relaxed, well supported and semi-flexed at elbow the tendons at elbow or wrist are sharply struck.
- 65**
The jaw reflex.
(Chart V) The patient's chin is firmly grasped with finger and thumb or a flat stick is placed in the patient's mouth resting on his lower teeth, the mouth being half open, and then the stick or the hand holding chin is struck sharply downward and the closure of the mouth noted.
- 66**
Kernig's reflex.
(Charts V & X) With thigh flexed at hip and leg flexed at knee, the patient either sitting or lying, the leg should be quickly extended at knee joint and a strong resistance to such extension noted, if present.
- 67**
Mechanical irritability.
Strike the nerve or muscle sharply with the finger or percussion hammer or press the nerve trunk or its tender points.
- 68**
Reinforcement.
The tendon, and to some extent the cutaneous reflexes, can be made stronger and can be often made to appear when apparently absent, by diverting the patient's attention in any way, usually by having him pull strongly on his clasped hands, his eyes being turned to the ceiling or to a picture at the instant the reflex is tested (Jendrassik).

CHART I d
Electricity, Lumbar Puncture, Brain Puncture, Ophthalmoscopy, Thermometry, Caloric Reaction

Comprising Numbers 70 to 80

METHODS OF EXAMINATION OF PATIENTS SUFFERING FROM NERVOUS DISEASES

ELECTRICITY AND LUMBAR PUNCTURE

METHODS OF TESTING

- 70
Faradism.
(Chart VII)
- The electrodes should be kept well moistened with warm salt water during the testing. The larger electrode is placed on sternum or back of neck or sacrum; while the smaller electrode, provided with an attachment for making and breaking (opening and closing) the current, is placed over the motor point of nerve or muscle. The secondary current of a faradic battery should be employed and the current should be gradually increased in strength by methods which vary in different batteries, until the faintest distinct contraction of the muscle occurs whenever the current is suddenly closed, the negative electrode being over the motor point. The test should be repeated several times. As the skin becomes moister a less strong current becomes necessary. It is important to make sure of the exact position of the motor point in each case by some preliminary tests and not to let the electrode slip away from this point during the testing.
- 71
Galvanism.
(Chart VII)
- With the electrodes arranged as above, first the negative, later the positive, electrode should be placed over the motor point of nerve or muscle and the strength of the current slowly increased by means of the rheostat until the faintest distinct contraction of the muscle occurs whenever the current is closed. The strength of the current causing this contraction, with each electrode in turn over the motor point, should quickly be read from the galvanometer, even before the needle has quite ceased its oscillations. In the same way read from the galvanometer the strength of the weakest current which will cause the faintest distinct contraction, when each electrode in turn is on the motor point and the current suddenly opened.
- 72
Muscle and
nerve.
(Chart VII)
- In all cases both the muscle and the nerve supplying it should be tested both by faradism and galvanism.
- 73
Character of the
contraction.
(Chart VII)
- Note the character of the muscular contraction, whether quick or sluggish (degenerative), or showing any peculiarity, and whether it is unusually persistent (myotonic), or whether it rapidly grows feebler under repeated tests (myasthenic).
- 74
Lumbar
puncture.
(Charts VIII &
XIX)
- The patient's body should be bent strongly forwards. Patient should, if possible, sit, but may be lying down. The skin having been thoroughly washed with alcohol, a horizontal line should be drawn from the posterior spine of one ilium to the other and a sterilized fine needle three or four inches long, preferably of platinum and with rather a short bevel, should be inserted between the laminae of the vertebrae immediately below or above this horizontal line. The needle may be inserted in the median line or a little to one side of it and pushed steadily forward and slightly upward until it enters the arachnoid sac when usually the cerebro-spinal fluid will escape in drops. If the needle be pushed too far it can be felt as it strikes the body of the vertebra and it should then be withdrawn about half an inch. It is rarely necessary and sometimes dangerous to attach a syringe and aspirate the fluid. If the needle becomes occluded clear it out with the stylet. It is better not to withdraw more than half an ounce of the fluid. Note the rapidity of escape, whether by drops or in a fine stream (tension), its appearance (cloudy, bloody, purulent). The fluid may be examined chemically (for albumen, sugar, cholin, etc.). A portion of the fluid, especially that containing the fine coagulum which frequently forms, is centrifuged, the clear fluid is carefully poured off and the bottom of the tube scraped and aspirated with a capillary pipette, the content of which is spread on a slide, fixed, stained and examined for cells (lymphocytes, leucocytes, bacteria, etc.). The cerebro-spinal fluid should also be tested for an increase of globulin, indicative of the presence of a syphilitic infection, of ancient or recent date, or of a meningitis, according to the method suggested by Noguchi (419). After lumbar puncture patients should remain quiet in bed during twenty-four hours. Even so, they are apt to suffer from headache, especially if much fluid has been withdrawn, or withdrawn too rapidly. Sometimes the nerve trunks of the cauda equina are injured, causing pain in the legs, but such pains are rarely severe and are of short duration. In some cases, in consequence of the withdrawal of the cerebro-spinal fluid, the medulla and cerebellum have been drawn down into the foramen magnum and death has resulted promptly. Such an accident is only possible in cases of cerebral tumor situated in the posterior fossa of the skull, and therefore lumbar puncture should not be performed in such cases.

BRAIN PUNCTURE, OPHTHALMOSCOPY, LARYNGOSCOPY, THERMOMETRY, AND THE CALORIC REACTION

- 75
Brain puncture This operation consists in trephining (with avoidance of the sinuses and large arteries) a small button from the scalp and bone, inserting a very thin needle canula and aspirating a small quantity of the brain substance, or tissue of a tumor, or fluid from a cyst. It has been many times performed and the results have been somewhat encouraging, but it is an operation which should be performed only by an experienced surgeon or neurologist and its detailed description is hardly in place here.
- 76
Ophthalmoscopy.
(Chart XIV) Examine the eyes for choked disc or optic neuritis, and for optic atrophy, retinitis, miliary tubercles, etc.
- 77
Laryngoscopy.
(Chart XIII) Examine the larynx for evidence of paralysis of one or more or of all its muscles.
- 78
Thermometry. It is often necessary to ascertain the temperature of the patient. The thermometer should be well washed in cool water both before and after taking the temperature. In taking the temperature in the mouth, the bulb of the thermometer should be placed well under the tongue and it should be noticed that the lips are held tightly closed during the two minutes that the thermometer is left in the mouth. In taking the temperature in the axilla, the axilla should first be wiped dry from sweat and care should be taken that the thermometer be surrounded by skin and not at all by clothes; the patient should be rolled over on his side in order to press arm firmly against chest and the thermometer should be left in position eight minutes. In taking the temperature in the rectum, a little vaseline or soap-suds should be put on the bulb before inserting it into the rectum, where it should remain two minutes. Instruments have been invented for taking the surface temperature of the skin of any part of the body, but they have not proved to be of much practical value.
- 79
Caloric
reaction.
(Chart XII) When one ear of a normal person, with head held upright, is syringed out with cool water there results a horizontal and rotatory nystagmus towards the other ear; when water warmer than the body is used, the nystagmus turns towards the syringed ear. This reaction does not occur in cases of destruction of labyrinth, or of paralysis of the vestibular nerve.
- 80
Cerebellar
nystagmus.
(Chart XXI;
1272) In lesions of the right cerebellar hemisphere, nystagmus to the right may only be seen, or may be made more marked, when the patient lies on the left side, and vice-versa.

CHART II
Analysis of the Subjective Symptoms of the Case
Comprising Numbers 81 to 190

ANALYSIS OF THE SUBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

List of diseases most likely to occur as the result of the etiological factors obtained from the history of the case.

81 Heredity, including con- sanguineous marriages in neuropathic families (Predisposing cause)	84 Inherited Diseases	Organic Diseases	<ul style="list-style-type: none"> 101 Idiocy and Imbecility 102 Spina Bifida and Meningocele 103 Hereditary (Huntingdon's) Chorea 104 Hereditary (Friedreich's) Ataxia 105 Myatonia Congenita 106 Myotonia Congenita (Thomsen's Disease) 107 Muscular Dystrophies 108 Syphilis of the Nervous System
	85 Inherited Tendencies	Neuroses	<ul style="list-style-type: none"> 109 Insanity 110 Epilepsy 111 Hysteria 112 Chorea 113 Neurasthenia 114 Neuralgia and Migraine. 115 Drunkenness (Alcoholism)
82 Personal Factors (Predisposing causes)	86 Age	Infancy and Childhood	<ul style="list-style-type: none"> 116 Cerebral Palsy of Childhood 117 Acute Anterior Poliomyelitis 118 Meningitis (tuberculous, etc.) 119 Hydrocephalus 120 Tetany And all the inherited diseases except 103 and 106
		Childhood and Youth	<ul style="list-style-type: none"> 121 Caries of Spine and Compression Myelitis 122 Meningitis (tuberculous, etc.) 123 Hereditary Ataxia 124 Glioma 125 Chorea 126 Epilepsy 127 Muscular Dystrophies 128 Hysteria 129 Insanity
		Adult	All other forms of Nervous Diseases and many of those above given
	87 Sex	More common in women	<ul style="list-style-type: none"> 130 Hysteria 131 Exophthalmic Goitre 132 Neuroses
		More common in men	<ul style="list-style-type: none"> 133 Locomotor Ataxia (Tabes) 134 Paresis 135 Injuries 136 Organic Diseases
	88 Race	Jewish & Latin	137 Neuroses
		Anglo-Saxon	138 Organic Diseases
	89 Dwelling Place, Habitation	Tropical	<ul style="list-style-type: none"> 139 Beri-Beri 140 Leprous Neuritis 141 Sleeping Sickness
		Dampness	142 Neuritis
	90 Occupa- tions	Overstrain	143 Occupation Neuroses
		Poisons	144 Neuritis

**83 Etiological
Factors
(Inciting
causes)**

91 Trauma- tism	Physical	{	145 Wounds and Injuries				
			146 Hemorrhage in Brain, Cord or Membranes				
			147 Meningitis				
			148 Myelitis				
			149 Disseminated Sclerosis				
			150 Neuritis				
			151 Tumors				
92 Poisons Toxic	{	Physical	152 Abscess				
			Psychical, Acute & Chronic	153 Hysteria			
				154 Insanity			
				155 Neurasthenia			
				156 Traumatic Neuroses			
			Metallic	157 Arsenical Neuritis			
				158 Lead Palsy, Colic, etc.			
159 Mercurial Tremor							
93 Infec- tions	{	Alcoholic	160 Multiple Neuritis				
			161 Neurasthenia				
		Tobacco, Tea or Coffee	162 Tremor				
			163 Neurasthenia				
		Narcotic	164 Drug Poisoning; Acute or Chronic				
			Germes and Toxines	{	165 Neuritis		
					166 Meningitis		
166 Myelitis							
167 Acute Anterior Poliomyelitis							
168 Landry's Paralysis							
169 Neuralgia							
170 Tetanus							
94 Syphilis	{	Tertiary Syphilis	171 Hydrophobia				
			172 Gumma				
			173 Meningitis Gummosa				
			174 Neuritis Syphilitica				
		Post-Syphilitic Infections	175 Endarteritis Syphilitica				
			176 Locomotor Ataxia				
			177 General Paresis				
95 Exhaust- tion	{	From Illness, Overstrain, Worry	178 Neurasthenia				
			179 Hysteria				
		From Venery and Masturbation	{	180 Neurasthenia			
				96 Extension of Inflam- mation	Caries of Skull or Vertebrae	{	181 Cerebral or Spinal Abscess
							182 Sinus Thrombosis
		183 Meningitis					
		184 Myelitis					
185 Neuritis							
97 Arterial Disease			186 Apoplexy				
98 Metastasis from Other Organs	{	187 Tumors					
		188 Tuberculous and Suppurative Meningitis					
99 Disease of Other Organs	{	Bright's Disease	189 Uremia				
			Diabetes Mellitus	190 Diabetic Coma			
		100 Cold is a doubtful direct, but probably an auxiliary etiological factor					

CHART III

Disturbances of Mental Activity

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the Symptoms of Disease.

200
Disturbances
of Mental
Activity.

201

CONSCIOUSNESS

The appreciation of one's existence and individuality as separate from the rest of the universe (Subject consciousness). The content of consciousness is the sum of the present perceptions of the various sensations (Object consciousness), together with the memories of past perceptions and judgments (Experience) (Chart XVI)

In disease, consciousness and intelligence may be either diminished or perverted as is set forth in Chart III a.

Neither intelligence nor consciousness is exaggerated or increased in disease, although the latter may be apparently so (Self-consciousness). In such cases, however, there is a concentration or limitation of consciousness rather than an increase of it; an exaltation of the subject, with a lowering of the object, consciousness.

202

INTELLIGENCE

The power of ascertaining facts and reasoning upon them. The power of discovering the relation of things and of acquiring knowledge (Chart XVI).

203

MEMORY

The power of retaining in the mind and of recalling at will perceptions and ideas formerly received. The more striking the perception and the more frequently it is repeated or recalled, the better becomes its memory (Chart XIII).

In disease, memory may be diminished in whole or in part, and the emotions may be either diminished or exaggerated as is set forth in Chart III b.

204

EMOTIONS

An emotion is a state of consciousness accompanied by a feeling of pain, pleasure, fear, anger, wonder, scorn, etc. In health a person's emotion is usually in harmony with his environment, but in disease it may be quite independent of the environment (Chart XVI).

Memory is never increased in disease, although certain memories may be accentuated and others lost.

CHART III a
Disorders of Consciousness and Intelligence
Comprising Numbers 201 and 202, and 205 to 218

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

CONSCIOUSNESS

		DIAGNOSTIC SYMPTOMS	DEFINITION	SIGNIFICANCE
201	D I M I N I S H E D	205 Coma	The patient lies in a profound stupor from which he cannot be aroused by irritation of any sensory organ (eye, ear, skin, mucous membrane, etc.). No voluntary acts are performed and the reflexes are abolished or diminished, except the circulatory and respiratory, which are often, but not always, deranged. Patient is unable to swallow. Lips and cheeks puff out during expiration.	These three conditions are not always sharply differentiated, but may merge into each other. They are due to loss or diminution of brain function in consequence of pressure upon the brain or of circulatory disturbances in it, or of poisons, etc. Occur in traumatism, and in many organic diseases of the brain and its membranes and especially of its blood vessels; also when toxic substances (morphia, etc.) or toxins (fever, etc.) are in the blood; also in Bright's disease and diabetes mellitus. Rarely the condition is functional.
		206 Semi-coma or Stupor	The patient is apparently in a coma but by strong sensory irritation can be aroused to some manifestation of consciousness. No voluntary acts are performed, but the reflexes are usually present. Patient can swallow. Patient may lie apparently awake, but really unconscious, with a low muttering delirium (Coma vigil).	
		207 Dazed, Bewildered, Somnolence or Sopor	The patient lies in a deep sleep or moves about automatically. Can be rather easily aroused, but does not fully appreciate his surroundings. Can speak more or less intelligently.	
		208 Erroneous personality	A mental condition in which a person imagines himself to be different from what he really is; sometimes an animal, sometimes a famous character in history, sometimes God, etc.	
		209 Double personality	At intervals the patient is in a sort of somnambulistic state and presents an abnormal consciousness and personality. His memory at times changes with his personality, in which case he remembers only occurrences in former similar conditions and not those of his normal state, and vice versa. This is a very rare condition and offers much opportunity for deception, and in some cases of hysteria may well be suggested by the examining physician.	
		210 Automatism Somnambulism	A person performs complicated and apparently intelligent acts, while suffering from loss, or great impairment, of consciousness, and retains little or no memory of the acts done.	
	P E R V E R T E D			Brain is probably anemic or exhausted, or the patient is under the influence of a great emotion (fright). Occurs in epilepsy, insanity, hypnotism, and rarely in hysteria (functional); not uncommon in childhood during sleep.

INTELLIGENCE

DIAGNOSTIC SYMPTOMS		DEFINITION	SIGNIFICANCE	
202 I N T E L L I G E N C E P E R V E R T E D	D I M I N I S H E D	211 Amentia	Absence or defect of intelligence, which is congenital or is acquired in infancy before the intelligence has developed.	Due to a malformed or diseased brain. Occurs in idiots, imbeciles and feeble minded persons.
		212 Dementia	Absence or defect of intelligence, which is acquired in later life in a person previously intelligent.	Due to atrophy or functional failure or diminution of blood supply of cerebral cortex. Occurs in insanity and is often its terminal stage.
		213 Hallucinations	Vivid perceptions of sensations (visual, auditory, olfactory, tactile, painful, etc.) not directly dependent upon any external corresponding reality; a sensation without an external object. They are usually regarded as real and are then associated with defective judgment and mental impairment, and therefore cannot be corrected.	
		214 Illusions	Erroneous perceptions. A false interpretation of an actual sensation, which is really of a different nature from that which the patient believes it to be. Frequently occur in rational persons, especially in those with defective terminal sensory organs. In such cases easily corrected.	
		215 Delusions	Erroneous judgments (often, but not always, dependent upon hallucinations) which can be corrected neither by reason, nor by the evidence of the senses and which are not in accord with universal human experience, and are the consequence of mental enfeeblement. Delusions are systematized or unsystematized according as they are supported or explained by more or less coherent reasoning, or not. The systematized delusions are of much more serious prognosis.	Due to disease of the cerebral cortex, whether functional, circulatory, toxic or organic. Usually symptoms of insanity, or of extreme degree of neurasthenia, are also present. In insanity these perversions of intelligence cannot be corrected by reason and demonstration, and in neurasthenia only rarely and imperfectly.
		216 Hypochondriasis	Delusions of imaginary symptoms and illness formed on an insufficient basis of abnormal sensations, which cannot be corrected and are associated with much mental depression.	
		217 Delirium	Irrational talk in persons with diminished consciousness. Probably due in most cases to hallucinations, illusions and mental confusion; consequently its irrationality may be in part only apparent. Often occurs in fevers.	
		218 Compulsory ideas and actions (275)	Certain thoughts or questions or doubts, which are forever in the patient's mind and cannot be removed. They may be of any nature. Patients are irresistibly compelled by an unknown force to do certain acts or to say certain words, usually quite trivial. Patients recognize the abnormal character of these ideas and acts and are made very unhappy by them, but are quite unable to prevent them.	

Methods for the detection of disorders of consciousness and intelligence are described in Chart I a.

For further discussion of these symptoms and the diseases in which they occur see Chart XVI.

CHART III b
Disorders of Memory and Emotions
Comprising Numbers 203 and 204 and 220 to 237

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

	DIAGNOSTIC SYMPTOMS	MEMORY	DEFINITION	SIGNIFICANCE
203 M E M O R Y D I M I N I S H E D	220 Amnesia		Inability to recall former perceptions and ideas. Loss of memory in general. May be more or less extensive. May affect memories of the immediate, or of the remote, past.	Functional or organic disease of the cerebral cortex, often anemia, sometimes the result of fright.
	221 Motor aphasia		Inability to express by words some idea in the patient's mind, although there is no paralysis of the vocal organs and the patient can usually express the idea by gesture. A loss of memory of how to speak (innervation memories), especially names. A limitation of the vocabulary.	Lesion in or near base of left inferior frontal convolution and anterior portion of left island of Reil in right handed persons, and of the right side in left-handed persons.
	222 Sensory or Auditory aphasia (word deafness)		Inability to understand (although not deaf) spoken words formerly intelligible. Loss of memory of words formerly heard. Hence inability to recognize them when spoken (233).	Lesion in or near posterior part of left superior temporal convolution and posterior portion of left island of Reil in right handed persons.
	223 Optic aphasia		Inability to name objects, which the patient sees clearly, although he can name them after feeling them. Loss of visual memories (232).	Lesion of left occipital lobe or of association fibers from this lobe in right handed persons.
	224 Mixed aphasia		A mixture of the three forms of aphasia just described.	Any one or a combination of the above lesions, or a lesion of the island of Reil, or of external capsule in right handed persons, in whom the above lesions are always in the left cerebral hemisphere, or, in slight degree, may result from carelessness.
	225 Paraphasia (Jargon speech)		The use of a wrong word, or the omission of a word, or the placing of the right word in the wrong place, in speaking, with consequent incoherent speech. Jargon speech is an extreme degree of this.	
	226 Paragraphia		The use of a wrong word, or the omission of a word, or the placing of the right word in the wrong place, in writing.	
	227 Agraphia		Inability to express in writing the idea in the patient's mind, although he formerly could do so and his right arm and hand are not paralysed.	Lesion in the base of the middle left frontal convolution, cortical or sub-cortical.
	228 Alexia (Word blindness)		Inability to read words patient could formerly read, although he sees them clearly and there is no paralysis of his vocal organs.	Sub-cortical lesion beneath left angular convolution in right handed persons.
	229 Astere-ognosis		Inability to recognize objects by the sense of touch, although there is no anesthesia present in sufficient degree to prevent it.	Lesion in or near cortex, or sub-cortex, of contralateral posterior central convolution.
	230 Apraxia		Inability to execute a desired act. Loss of skill in executing acts, although there is no motor paralysis present. Loss of innervation memories necessary to perform these acts.	Cortical, or sub-cortical, lesion of motor area of contralateral hemisphere.
	231 Agnosia		Inability to recognize objects through some organ of sense which is itself normal. This may be due to failure of full perception or to loss of special memories.	Cortical, or sub-cortical, lesion of sensory area of cortex of contralateral cerebral hemisphere.
	232 Psychic blindness		Inability to recognize well known objects or to comprehend familiar things by sight, although the patient is not blind. Loss of visual memories, optic aphasia (223).	Cortical, or sub-cortical, lesion of left occipital lobe, except in region of calcarine fissure.
	233 Psychic deafness		Inability to recognize and comprehend well known words and sounds, although the patient is not deaf. Loss of auditory memories. Includes sensory aphasia (222).	Cortical, or sub-cortical, lesion in left superior temporal convolution in right handed persons.

DIAGNOSTIC SYMPTOMS		EMOTIONS	SIGNIFICANCE
		DEFINITION	
204 E M O T I O N S	{ E X A G G E R A T E D D I M I N I S H E D	234 Sadness (Melan- cholia)	Without adequate cause the patient is depressed and unhappy. There is a great repression of mental and physical activity usually. He can be influenced little, if at all, by reason; difficult to get his attention.
		235 Fear (Phobias)	Without adequate cause the patient is in constant fear of an impending calamity, or has an unformulated fear. He dreads to cross an open space (agoraphobia), or to enter a small room or confined space (claustrophobia), or fears a storm (astrophobia), or syphilis (syphilophobia), or ill-timed urination (urophobia), or everything (pantophobia), etc. Can be influenced little, if at all, by reason. Frequently has a more or less unconscious sexual basis.
		236 Joy (Mania)	Without adequate cause the patient is exhilarated. There is great exuberance of mental and physical activity. Careless and destructive. Can be influenced little, if at all, by reason. Difficult to get his attention.
		237 Apathy	Without adequate cause patient is in a dull stuporous condition. No expression of physical or mental activity. An automaton, submitting passively to whatever is done for him.
			Functional or circulatory disturbance of cerebral cortex, especially cerebral exhaustion. Occurs in neurasthenia and especially in insanity.
			Fears and apprehension seem to be the basic symptoms of many forms of incipient insanity (Mosher).

Methods for the detection of disorders of memory and emotion are described in Chart I a.

For further discussion of these symptoms and of the diseases in which they occur see Charts XIII and XVI.

CHART IV

Disorders of Voluntary Motion

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)
Definition, Significance and Relationship of the Symptoms of Disease.

240 DISORDERS OF VOL- UNTARY MOTION

The power of executing movements by an effort of will is acquired in early life. The process is quite obscure, but seems to depend upon the existence of innervation memories of past acts, primarily reflex. Voluntary motion depends upon the integrity of the central motor neurons (461) and of the peripheral motor neurons (462). In disease the power of voluntary motion may be diminished, exaggerated or perverted.

MUSCULAR TONICITY

Closely connected with the power of voluntary and involuntary action is the fact that the muscles of a normal person are in a condition of constant, slight, but varying, contraction. This is called muscular tonicity or tone. It is really a reflex act caused and maintained by many slight irritations, and can be abolished by cutting the posterior nerve roots. Muscular tonicity is increased (hypertonia) in destructive lesions of the central motor neurons and in some functional disorders. It is diminished (hypotonia,) or abolished (atonia,) in destructive lesions of the peripheral motor or sensory neurons, in lesions of the cerebellum, in sleep and in narcosis.

241 DIMINUTION also called AKINESIS and HYPOKINESIS

242 EXAGGERATION also called HYPERKINESIS

243 PERVERSION also called PARAKINESIS

244 PARALYSIS

A condition in which the muscles cannot be contracted by the strongest effort of the will. As commonly used the term includes:

PARESIS

A condition in which the muscles can be contracted only feebly by the strongest effort of the will.

The conditions under which paralysis or paresis occur are set forth in Chart IV a.

245 TONIC SPASM

A continuous, involuntary, muscular contraction of longer or shorter duration (572).

246 CLONIC SPASM

More or less rhythmical alternations of involuntary, coarse, violent muscular contractions and relaxations (571). Must not be confounded with a coarse tremor.

The conditions under which the various forms of spasm occur are set forth in Chart IV b.

247 IRREGULAR SPASM

Involuntary acts of various kinds (292, 573-4).

248 ATAXIA

Disorderly movements due to loss of power of co-ordination (638). Asynergia. Associated with hypotonia (252)

249 LOSS OF SKILL, APRAXIA

Awkwardness.

The conditions under which the various forms of perversion of motion occur are set forth in Chart IV c.

250 TREMOR

Involuntary rhythmical oscillation of some part of the body or of a muscle. Less powerful, more rapid and more rhythmical than a clonic spasm but similar in appearance, especially when coarse. Tremor may be slow (3 to 6 per second) or rapid (8 to 12 per second). It may be coarse or fine (639).

CHART IV a
Motor Paralysis

Comprising Numbers 244 and 251 to 260

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

244 PARALYSIS { CHARACTER
EXTENT

MOTOR PARALYSIS

		DIAGNOSTIC SYMPTOMS	DEFINITION	SIGNIFICANCE
P A R A L Y S I S	{ C H A R A C T E R	251 Spastic, or hyper- tonic, paralysis. (473). (Figs. 24-6)	A paralysis in which the muscles show increased tone and offer much resistance to passive motion, especially rapid motion. The normal excursion of the joint is restricted. The muscles have their normal volume and under the microscope their fibers show a normal appearance. The electrical reaction of muscle and nerve is normal (396). The tendon reflexes are increased.	Destructive lesion of central motor neurons (461). It occurs in diseases of the brain or spinal cord, or may be functional. Rarely a reflex spasm (268), especially preputial irritation in children, or pain, may simulate this condition.
		252 Flaccid, or hypo- tonic, or atonic, or atrophic paralysis (472). (Figs 24-6)	A paralysis in which the muscles have lost their tone and offer little or no resistance to passive motion, even when rapid. The joint has a normal or even increased excursion. The muscles exhibit a great and rapid atrophy, and under the microscope their fibers show a loss of their transverse striation and various forms of degeneration (fatty, hyaline, etc.). The electrical reaction of degeneration is present (399). When muscles are completely degenerated (404) passive contractures (263) may occur. The tendon reflexes are abolished or diminished.	Destructive lesion of peripheral motor neurons (462). It occurs in diseases of the muscles, peripheral nerves, anterior horns of cord, or motor nuclei in brain stem. It is never functional, but may be somewhat simulated by joint disease. Hypotonia without muscular paralysis or atrophy occurs in cerebellar lesions, tabes and other ataxic conditions (240).
		253 Myasthenic paralysis (553)	A rapid tiring of muscles upon exercise. A myasthenic reaction to electricity (401). Muscles show small foci of small round cells.	A lesion of the muscles and often of thymus gland.

MOTOR PARALYSIS (Continued)

DIAGNOSTIC SYMPTOMS

DEFINITION

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254 Hemiplegia (478-9) (Figs. 17-24)	A paralysis with exaggerated tendon reflexes, of one lateral half of the body and extremities limited by the median line in front and behind. It is partial, if limited to arm and leg; complete, if arm, leg, tongue, palate and face are all involved. In some cases of hemiplegia there are slight weakness and exaggerated reflexes on the other side of the body also, especially in the leg. Symmetrical, bilateral muscles, which have a common function and a bilateral cortical innervation, are not paralysed; at most temporarily weakened. Such are the ocular, masticatory, laryngeal, respiratory, bladder, rectal, etc., muscles. In cerebral hemiplegia certain muscles are, in most cases, more completely paralysed than others. These "predilection muscles of Wernicke" are the trapezius, the external rotators and adductors of the upper arm, the triceps, the supinators and abductors of thumb, the extensors of the thigh, the flexors of the leg and the dorsal flexors of the foot.	A lesion of the contralateral central motor neurons (461). In extremely rare cases the lesion may be homolateral (homolateral hemiplegia), in which cases the pyramidal tract may not decussate in the medulla. Hemiplegia is usually due to a cerebral lesion, but the partial form may be due to a bulbar or spinal lesion, very rarely. Very rarely, there may be no lesion, except an extreme local anemia or edema of brain as in nephritis (hemiplegia sine materia).
255 Diplegia (478)	A double hemiplegia involving both sides. May be complete or partial and not infrequently is limited to the legs, or the face (facial diplegia), etc.	A lesion, usually but not always cortical, of the central motor neurons or basal nuclei on both sides.
256 Crossed paralysis (537-42) (Hemiplegia alternans) (Figs. 20-1)	A paralysis of one or more homolateral cranial nerves and of the contralateral arm and leg.	Always due to a lesion involving the pyramidal tract with other structures in the brain stem (460); either in the medulla (hypoglossal hemiplegia alternans (1268)), the pons (facial hemiplegia alternans (1269)), or in the crus cerebri (motor oculi hemiplegia alternans (1270)). The nuclei, or the neurons, peripheral or central, of the cranial nerves are involved below the decussation of their central neurons.
257 Paraplegia (480) (Figs. 24-6)	A symmetrical paralysis of both sides of the body. Usually only involves the legs and lower part of body, but may involve the arms and even both sides of the face.	May occur in lesions of the muscles (dystrophies) (477), or of the peripheral nerves (neuritis (488-9)), or of the spinal cord or brain stem, or even of the cerebral cortex (bilateral lesion). The distinction between paraplegia and diplegia (255) is not always sharply drawn. In general diplegia is applied to paralysees of cerebral origin, paraplegia to those of spinal or peripheral origin.
258 Monoplegia (479) (Fig. 15)	A paralysis of one extremity only, or of one half of the face only.	May be due to lesion of motor cerebral cortex, or of the motor nuclei, or of the peripheral nerves.
259 Local paralysis (481) (Fig. 15)	A paralysis limited to one or more muscles of the face, eye, mouth, neck, body or extremities. Less than a whole extremity.	May be due to lesions of muscles or of peripheral nerves, or of spinal cord, or rarely of motor cerebral cortex, or functional.
260 Aphonia (737-8)	Inability to produce vocal sounds. Absence of voice, but whispering is possible.	A variety of local Laryngeal paralysis, organic or functional.

Methods for the detection of paralysis and paresis are described in Chart I b.
For further consideration of these symptoms and of the diseases in which they occur, see Chart X.

CHART IV b

Spasm

Comprising Numbers 245 to 247 and 263 to 276

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

SPASM

	DIAGNOSTIC SYMPTOMS	DEFINITION	SIGNIFICANCE
245 T O N I C S P A S M	263 Passive contracture (Figs. 24-6)	A continuous contraction of long duration in which the muscles, tendons and ligaments have become anatomically shortened and cannot be extended by force, even under etherization. The muscle fibers are degenerated, while the connective tissue of the muscle is hypertrophied and usually secondarily contracted, as in other newly formed connective, or scar, tissue.	Due to muscular lesions and to degeneration of the peripheral motor neurons (462).
	264 Active contracture (Figs. 15, 17, 24-6)	A continuous contraction lasting weeks, months, or years, which can be overcome by force, either with or without etherization. Muscles are in a normal condition of nutrition. Most common in the arms, or legs, or neck muscles (torticollis). The active contracture of a hemiplegia is usually that of flexion in the arm and of extension in the leg.	Active contractures occurring in hemiplegia affect the muscles not absolutely paralyzed. When the contracture is overcome by the application of a plaster of Paris splint, the muscles often show a surprising degree of voluntary motion, when the splint is removed. These contractures depend in part, on attempts at voluntary movements and on associated movements, but in greater part on reflex action from sensory irritation; the inhibitory action of the brain being cut off by the lesion. They never occur in hemiplegia in tabetics and in any case can be relieved by section of the posterior nerve roots. Such contractures are always of very bad prognosis as to recovery.
	265 Myotonia (613)	An active contracture of brief duration but much longer than a convulsive tic. It may occur at the commencement of voluntary motion (Thomson's disease, or myotonia congenita) or may be excited by cold (Eulenberg's disease, or paramyotonia congenita). It is frequent in meningitis and tetanus in which it takes many forms, viz: "retraction of head;" "trismus," strong closure of jaw; "opisthotonus," arching of body backwards; "pleurosthotonus," bending of body to one side; "emprosthotonus," arching of body forwards and "orthotonus," holding of body rigid and straight.	Active contracture is sometimes due to paralysis of antagonist muscles or to muscle lesions.
	266 Rigidity	An active contracture of such mild degree that it does not prevent passive, or even voluntary, motion of the part, although rendering it difficult (paralysis agitans (612)), etc.	All tonic spasms (not including passive contracture) are due to a functional disorder, or are reflex (especially in children), or are due to irritation (chemical, sensory or vascular) of central motor neurons (461).
	267 Convulsive tics (601)	A violent spasm of momentary duration. If rapidly repeated it must be classed under myoclonus (270 and 601-4). If painful, it is called "tic douloureux" (602).	Painful cramps, especially in legs, of the nature of myotonia or tics, may be due to a deficiency of water in the system.
	268 Reflex spasm	A spasm, usually tonic, caused by irritation of some sensory tissue.	

SPASM (Continued)

DIAGNOSTIC SYMPTOMS		DEFINITION	SIGNIFICANCE
246 C L S O P N A I S C M	269 Convulsion (571)	Violent clonic contractions of many, or of all the, muscles of the body.	Clonic spasms are usually due to irritation of the cerebral cortex, but may also result from very exaggerated reflexes (clonus).
	270 Myoclonus or convulsive tics	Successive clonic contractions of one, or of a few adjacent muscles. Repeated convulsive tic. Most common in the face muscles (blepharospasm (601)).	
	271 Athetosis or mobile spasm (574)	Slow, worm-like, rhythmical movements, often associated with transitory contractures (spasmus mobilis), of fingers and wrists and more rarely of toes and ankles. Hyperextension is the predominant action. Usually unilateral, but may be bilateral. Much more common in children than in adults. Muscles of the neck, face and of other parts of the body are not infrequently involved.	
247 I R R E G U L A R S P A S M S	272 Choreic move- ments Chorea minor (573)	Rapid, irregular, co-ordinated, but purposeless movements caused by contraction now of one group of muscles, now of another, throughout the body; bilateral or unilateral (hemichorea). Cease during sleep. They often render voluntary movements ataxic and are usually associated with a mild degree of paralysis of the muscles involved.	Lesion is usually in posterior part of optic thalamus or corpus striatum of contralateral hemisphere and not causing complete paralysis. Lesion may involve the fibers connecting the optic thalamus with the cerebral cortex. May occur in diffuse cortical lesions.
	273 Chorea major or magna (628)	Patient performs involuntarily and uncontrollably a complicated and apparently purposeful movement. Also applied to a coarse tremor or violent oscillation of a part of the body.	
	274 Habit chorea (626)	Patient frequently performs involuntarily, and usually unconsciously, the same act. Usually a small act.	
	275 Compulsory acts (218)	Patient is compelled by some power within him which he cannot understand or explain to perform certain acts against his will.	Functional disorders, occurring in the neuroses and in insanity.
	276 Associated move- ments	Muscular contractions, occurring when movements are executed or attempted, in muscles not directly concerned in the movement attempted; often the corresponding muscles of the opposite side of the body, often those of the face. Such associated movements are Bell's phenomenon (444), Strümpell's tibialis phenomenon (445), Babinski's associated movements in unilateral paralysis (446).	

Methods of detection of spasm are described in Chart I b.

For further discussion of these symptoms, and of the diseases in which they occur, see Charts XI and XII.

CHART IV c
Perversions of Motion
Ataxia, Loss of Skill, Tremor
Comprising Numbers 248 to 250 and 280 to 293

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

ATAXIA—LOSS OF SKILL

DIAGNOSTIC SYMPTOMS		DEFINITION	SIGNIFICANCE
248 A T A X I A	280 Motor ataxia (644) (dynamic ataxia) (Figs. 24-6)	Voluntary movements are executed in an irregular and disorderly manner, which is due to a loss of the co-ordinating power. Rarely associated with decided vertigo.	Is due to a loss of muscle sense (42) (deep sensibility). May be due to lesions of peripheral sensory nerves, or of posterior columns of cord, or of brain stem, or of cerebral cortex posterior to fissure of Rolando, or may be toxic (alcohol), or functional.
	281 Cerebellar ataxia (642) (static ataxia) (Figs. 19-26)	Walking and standing are inco-ordinate, but other acts are not, or only slightly so. Patient executes simple movements of his legs fairly well when lying in bed, but in walking and standing he lacks synergy of the muscles and staggers and sways like a drunken man. Usually associated with vertigo (392).	Is due to a lack of muscular synergy (41) (asynergy). Due to lesion or functional disorder of the cerebellum or its tracts, including the direct cerebellar tract in brain stem or cord, or to tumors in frontal lobe of brain, or to disease of ears or eyes, or to poisons (alcohol, etc.). In lesions of the cerebellar hemisphere the disorder is transitory; in lesions of the worm it is more permanent.
249 L O S S O F S K I L L	282 Apraxia (Fig. 15)	Inability, or difficulty, in performing a desired and accustomed act because of loss, or derangement, of the innervation memories concerned in that act. Loss of skill.	Loss of innervation memories, general or partial, due to cortical or sub-cortical lesions (anterior or posterior central, or supra-marginal convolution) or to functional or anemic disorders of cerebral cortex. (See page 26-7).
	283 Anarthria (737)	Absence of speech. Speech may never have been acquired, as in idiocy, or it may be voluntarily restrained for a purpose; or it may be more or less involuntarily restrained, as in insanity or hysteria.	May be either functional or organic and, if the latter, may or may not be due to lesions in the organs of speech. If not, it is called pure motor aphasia or aphemia.
	284 Dysarthria (738)	Such difficulty in articulation that speech becomes indistinct and blurred, but is probably never so great as to cause complete anarthria (283).	Occur in lesions of the medulla and pons (bulbar paralysis, Figs. 21-3) and of the cranial nerves. Also in diphtheria, hydrophobia, myasthenia gravis, rarely in trichinosis and frequently in hysteria (globus hystericus).
	285 Dysphagia	Difficulty in swallowing.	
	286 Dysmasesis (553)	Difficulty in mastication.	
	287 Astasia and Abasia (653 and 792)	Complete inability to stand or walk but legs can be moved freely, even strongly, when lying or sitting.	A delusion or auto-suggestion, which occurs in hysteria. May occur rarely in cerebellar lesions.
	288 Diadocokinesis (36)	Difficulty in repeating a movement rapidly, especially supination.	Occurs in lesions of a cerebellar hemisphere, or is functional.

TREMOR

250 T R E M O R	DIAGNOSTIC SYMPTOMS	DEFINITION	SIGNIFICANCE
	289 Passive tremor (646 and 647)	Involuntary, rhythmical oscillation or trembling of a part which is otherwise at rest.	Functional. Occurs in paralysis agitans, weakness, etc.
	290 Intention tremor (645)	An involuntary tremor which only occurs when a voluntary motion is made, or is willed and is about to be made.	Functional and organic. Occurs in neuroses and in organic diseases (disseminated sclerosis)
	291 Nystagmus (640)	An involuntary trembling or oscillation of eyeball, usually horizontal, rarely vertical, very rarely rotatory. Increased, or only occurs, on voluntary motion of eyeball, especially on extreme deviation. The rapidity of the oscillations varies from 60 to 200 per minute. Their amplitude from 2 to 4 millimeters. Nystagmus may be oscillatory when the motion in each direction is equally rapid, or rhythmic when it is quicker in one direction than in the other.	Occurs especially in lesions of the vestibular and other nuclei in the pons, Deiter's nucleus in the cerebellum, the posterior longitudinal bundle in the brain stem, in disturbances in the semi-circular canals, and in weakness of ocular muscles, and in lesion of ponto-cerebellar angle; also in the caloric reaction (79), and in cerebellar disease (80).
	292 Fibrillary contraction or fibrillation (641)	An involuntary contraction of a bundle of fibers of a muscle of short duration. When many occur in adjacent bundles at short intervals, waves of contraction run over the muscle, but do not cause it to contract as a whole.	Degeneration of those multipolar nerve cells in the anterior horns of the spinal cord and brain stem of which the motor nerves supplying the muscle are the axons. Rarely occurs in traumatic neuroses.
	293 Myokymia (697)	A fibrillary twitching of the muscles occurring in healthy persons.	Normal. Exhaustion. Following excessive muscular contraction or exposure to cold.

Methods of detection of perversions of motion are described in Chart I b.

For the further discussion of these symptoms and of the diseases in which they occur, see Chart XII.

CHART V

Reflex Activity

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the symptoms of disease.

296 REFLEX ACTS

An involuntary movement caused by irritation of a sensory nerve or terminal organ. Although not the result of conscious intention, yet these acts seem purposeful and usually tend towards the protection of the body. In order that a reflex act may take place there must be a comparatively healthy reflex arc, consisting of a motor nerve, a sensory nerve and some gray matter connecting the two; or, in other words, a motor neuron and a sensory neuron connected together directly or by a bridging neuron. Reflex acts are inhibited and modified by inhibitory impulses passing down from the brain along the so-called inhibitory fibers, which are also the central motor neurons (the pyramidal tract) (472-4, 810). (Figs. 19, 24).

297 CUTANEOUS OR SUPERFICIAL REFLEXES

A reflex act which originates from an irritation of the skin (57).

298 MUCOUS MEMBRANE REFLEXES

A reflex act which originates from an irritation of a mucous membrane (58).

299 TENDON OR DEEP REFLEXES

A reflex act which originates from the sudden stretching of the fibers of a muscle (60-6).

300 ORGANIC REFLEXES

A reflex act affecting one of the viscera of the body (1), especially the bladder or rectum.

301 VASO-MOTOR REFLEXES

A reflex act affecting the arterioles (59).

302 PUPILLARY REFLEX

A reflex act affecting the pupil (25-7).

The conditions in which reflex acts are disordered are set forth in Chart V a.

The conditions in which the pupillary reflexes are disordered are set forth in Chart V b.

CHART V a

**Cutaneous or Superficial Reflexes, Mucous Membrane
Reflexes, Tendon or Deep Reflexes, Organic
Reflexes, Vaso-Motor Reflexes**

Comprising Numbers 303 to 326

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

CUTANEOUS REFLEXES

DIAGNOSTIC SYMPTOMS	DEFINITION AND LOCATION OF REFLEX CENTERS	SIGNIFICANCE
303 Plantar	Plantar flexion of the toes when the sole of the foot is irritated. (1st and 2nd sacral segments.)	The abnormal reflexes, Babinski, Gordon and Oppenheim reflexes and ankle-clonus, always indicate disease of the central motor neurons (461), except in infants, in whom these reflexes (except ankle-clonus) may be present normally, and in some cases of hysteria in which an imperfect ankle-clonus may rarely be obtained. The Babinski reflex is most reliable in a diagnostic sense. The Oppenheim reflex is sometimes present when the Babinski is absent and vice versa. Kernig's sign indicates meningitis or meningismus; it is an important, but not a certain, diagnostic sign.
304 Babinski's	Sluggish extensive dorsal flexion of the great toe when the sole of the foot is irritated.	
305 Gordon's	Dorsal flexion of the great toe when deep pressure is made through the calf muscle on the deep flexor muscles beneath; the leg being completely relaxed.	
306 Oppenheim's	Dorsal flexion of the great toe elicited by firm stroking with a hard object, or finger, just behind the postero-internal border of the tibia from above downwards; the leg being completely relaxed.	Alterations in the tendon reflexes are of very much greater diagnostic value than are those of the cutaneous (except the Babinski) reflexes, which are in many cases inconstant, probably because the cutaneous reflex impulses may even pass through the gray matter of the brain (cerebellum) as well as through a wide area of that of the spinal cord.
307 Gluteal	Contraction of the buttocks when the skin covering them is irritated. (4th and 5th lumbar segments.)	Diminution of reflexes is usually of little diagnostic value, but their abolition is of great value and may be due to a destructive lesion of any part of the reflex arc (a peripheral motor neuron, a peripheral sensory neuron, or a central bridging neuron). When there is a lesion of the peripheral motor neuron, atrophic motor paralysis is present in addition to the loss of the reflex. When there is a lesion of the peripheral sensory neuron there is usually a sensory paralysis (anesthesia, etc.), in addition to the loss of the reflex. Diminution or abolition of reflex activity (cutaneous or tendon) may occur, at least temporarily, in acute diseases or other forms of irritation of the central motor neurons; also in cases of shock, exhaustion, coma, narcotism and after epileptic fits, (except Babinski); also by will power and by voluntary movements of the muscles concerned; also (except Babinski) in cases of complete separation of the brain from the spinal cord, and, rarely, of increased intracranial pressure, also frequently in fevers.
307a Anal	Contraction of sphincter ani upon pin pricks of anus. (5th sacral segment.)	
308 Cremasteric	Drawing up of the testicle when the inner side of the thigh is irritated. (1st to 3rd lumbar segments.)	
309 Umbilical	Sudden movement of umbilicus towards the side of abdomen irritated. (8th to 12th dorsal segments.)	
310 Epigastric	Sudden retraction of epigastrium when the hypochondrium is irritated. (7th to 9th dorsal segments.)	
311 Interscapular	Drawing inwards of the scapula when the skin of the interscapular space is irritated. (5th cervical to 1st dorsal segments.)	
312 Corneal or conjunctival	Closing of the eyelids when the cornea or conjunctiva is irritated. (5th to 7th cranial nuclei.)	
313 Nasal	Sneezing when the nasal membrane is irritated. (5th to 10th cranial and upper cervical nuclei.)	The abolition of the knee-jerk is of great diagnostic importance. It is absent in tabes, neuritis (multiple and crural), acute anterior poliomyelitis involving the extensor cruris, Landry's paralysis, lesion of the cauda equina or of the lumbar enlargement, during the attack of family periodic paralysis, when exhausted after an epileptic attack and in cases of muscular dystrophy involving the extensor cruris muscles. It is usually abolished in Friedreich's ataxia and combined sclerosis except in the early stages when it may be increased. It may be absent in cerebral compression (tumor or meningitis) and in some cases of cerebellar disease, and may then be unilateral. It may be absent also in the conditions mentioned in the preceding paragraph.

MUCOUS MEMBRANE TENDON, ORGANIC AND VASO-MOTOR REFLEXES

DIAGNOSTIC SYMPTOMS	DEFINITION AND LOCATION OF REFLEX CENTERS	SIGNIFICANCE
314 Uvular	Raising of the uvula in phonation or upon irritation of its mucous membrane. (9th to 10th cranial nuclei.)	Exaggeration of the reflexes may be due to a mild inflammation, or to any irritation, of any part of the reflex arc. Strychnine increases reflex activity by irritating the nerve cells in the anterior horns. More commonly the reflexes are increased by any lesion of the motor central neurons, thus cutting off the normal inhibitory influence of the brain, and are then associated with paralysis of voluntary motion. The presence of ankle-clonus, the Babinski reflex and the dorsal foot reflex indicates a lesion of the pyramidal tract much more certainly than does an exaggerated knee-jerk, unless the latter is associated with an adductor contraction. Very commonly the reflexes are increased in functional diseases (hysteria) and in nervousness.
315 Pharyngeal	Retching or gagging when the pharynx is irritated. (9th to 10th cranial nuclei.)	
316 Ankle-clonus	Oscillation of the foot when the ball of foot is pressed quickly and continuously upwards. (5th lumbar and 1st sacral segments.)	
317 Achilles reflex	Sudden plantar flexion of foot when the tendo-Achillis is sharply struck. (1st to 2nd sacral segments.)	
318 Knee-jerk	Sudden extension of knee when the ligamentum patellae is sharply struck. When this reflex is exaggerated it is usually accompanied by a contraction of the adductors of the opposite thigh, or even by knee clonus (61). (2nd to 4th lumbar segments.)	Innervation of the muscles not concerned in the reflex act and diverting the attention increases reflex activity (reinforcement, 68).
319 Kernig's sign	Resistance to sudden extension of the knee.	The paradoxical reflex is of no diagnostic importance. It consists in a contraction of the tibialis instead of the calf muscles when ankle-clonus is tested for; also of a contraction of the flexors instead of the extensors of the thigh when the knee-jerk is tested for.
320 Dorsal foot reflex	Sudden plantar flexion of the toes when the dorsum of the foot over the 4th and 5th metatarsal bones is struck. (5th lumbar and 1st sacral segments.)	
321 Elbow and wrist reflexes	Sudden extension or flexion of elbow or wrist when the corresponding tendons are sharply struck. (5th to 7th cervical segments.)	In the dorsal foot-reflex (Mendel-Bechterew) normally there is either no reflex or a dorsal flexion of the toes, but in cases of pyramidal tract lesions a plantar flexion of the toes occurs.
322 Maxillary reflex	Sudden closure of jaw when it is sharply struck downwards. (5th cranial nucleus.)	Inability to void urine, or to retain it, is sometimes due to nervousness and sometimes to mechanical obstruction (enlarged prostate or stricture), but any other serious disturbance of the organic reflexes indicates organic disease of the nervous system. It never occurs in diseases limited to the peripheral nerves, except in lesions of the cauda equina, and rarely in cerebral disease. It is most common in spinal disease, sphincter paralysis with empty bladder and constant dribbling of urine in lesions of lumbar enlargement, and detrusor paralysis with distended bladder and often with dribbling of urine in lesions above the lumbar enlargement. (Fig. 28.)
323 Bladder or vesical reflex	The retention of urine in the bladder by the sphincter reflex, and the expulsion of urine by the detrusor reflex and the synchronous relaxation of the sphincter. (Hypogastric sympathetic ganglia.)	
324 Rectal reflex	Similar to that of the bladder. (Hemorrhoidal sympathetic ganglia.)	
325 Ischemic reflex	A sudden pallor of the skin following an irritation and limited to the area of irritation.	
326 Paralytic, hyperemic reflex (dermographia)	Congestion of the skin following the ischemia due to irritation; (tâches cérébrales and dermographia).	Vaso-motor disturbances cause a disturbance of the nutrition of a part. Diseases which result from, or are associated with, disturbances of the vaso-motor reflexes are discussed in Chart XVII.

The methods of eliciting the various reflexes are described in Chart I c.

Diseases in which the reflexes are altered are discussed in Charts X, XIV, XVI, XVII.

CHART V b

Pupillary Reflexes

Comprising Numbers 302 and 330 to 341.

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

PUPILLARY REFLEXES

	DIAGNOSTIC SYMPTOMS	DEFINITION AND LOCATION OF REFLEX CENTERS		SIGNIFICANCE
302 P U P I L L A R Y R E F L E X E S	330 Pupillary reaction to light (25)	Pupil contracts when light is thrown on retina of the same eye (direct reflex), and when light is thrown on retina of opposite eye (consensual reflex), and dilates when retina is shaded from light (ciliary ganglion).		The pupillary reaction to light is diminished or absent in lesions of the reflex arc (optic nerve, corpora quadrigemina, the Westphal-Edinger cell group of the motor oculi nucleus, third nerve and ciliary ganglion), especially in lesions of the ciliary ganglion. When the optic nerve or corpora quadrigemina are involved the consensual reflex can not be obtained from the other (healthy) eye. It is absent in blindness, deep sleep, narcosis, shock, coma, epileptic, and occasionally in hysterical, attacks; also absent in tabes, in many cases of paresis and in rare cases of syphilis alone; absent also when eye is under the influence of mydriatics or myotics.
	331 Pupillary reaction to accom- modation (27)	Pupil dilates when patient looks at a distant object and visual axes are parallel and contracts when patient looks at a near object and eyes converge.		The pupillary reaction to accommodation is absent (cycloplegia) in lesions of the third nerve, sometimes after diphtheria, occasionally in alcoholism and when the eye is under the influence of mydriatics or myotics, also in myopia and in cases of deficient convergence.
	332 Argyll- Robert- son's phe- nomenon (447, 891)	Pupil does not respond to light, but does respond to efforts at accommodation.		The Argyll-Robertson's phenomenon occurs in almost all cases of tabes and paresis (in many of these cases a degeneration of the posterior columns of the cord has been found at autopsy) and very rarely in cases of syphilis in which there are no manifestations of either tabes or paresis for years afterwards. The reverse of the Argyll-Robertson's phenomenon, i.e., the preservation of the light reflex and the loss of the accommodation reflex, occurs occasionally in diphtheritic paralysis and has been found associated with syphilis, basal meningitis, tumors of corpora quadrigemina and myelitis. It is extremely rare.
	333 Immobile pupil (545)	The pupil responds neither to light nor accommodation, but in some cases may still dilate slightly on irritation of cervical sympathetic.		Immobile pupil may occur in lesions of the optic nerve or tract or in its nucleus or in that of the third nerve or in the ciliary ganglion or its nerve. It may also be associated with ophthalmoplegia externa or interna or both. When it occurs alone it is due to a lesion in the nucleus. Immobile pupil also occurs in tabes, in epilepsy, in some forms of hysteria, in fainting, and in katatonic stupor.
	334 Hemiopic reflex (26)	Pupil contracts when light is thrown on the unparalysed half of retina, but does not contract when light is thrown on paralysed half.		The hemiopic reflex occurs only in lesions of the optic tract or geniculate bodies (homonymous hemianopia) or of the central part of the optic chiasm (bitemporal hemianopia). The existence of this reflex is disputed by many observers.

PUPILLARY REFLEXES (Continued)

	DIAGNOSTIC SYMPTOMS	DEFINITION AND LOCATION OF REFLEX CENTERS	SIGNIFICANCE
P U P I L L A R Y R E F L E X E S (C o n t i n u e d)	335 Cilio- spinal reflex (465, 1191-2)	Pupil dilates when neck on same side is irritated or when cocaine is dropped in the eye. (Cer- vical sympathetic ganglion.)	The cilio-spinal pupillary reflex is absent in lesions of the cervical sympathetic, and in many lesions of the medulla and lower cervical and upper dorsal region of the spinal cord (cilio-spinal center—465). Hippus is usually associated with a general exaggeration of reflexes. Westphal's pupillary reaction occurs in some cases of tabes and in paresis.
	336 Hippus	When the eye is sud- denly exposed to light, there occurs a series of alternate contractions and dilatations of the pupil, gradually growing less in degree.	The paradoxical pupillary reflex is of no diagnostic sig- nificance. It has been observed in tabes and in paresis and is the result of fatigue. Mydriasis may be irritative or spasmodic, due to irrita- tion of the cervical sympathetic ganglion or nerve; or may be paralytic, due to paralysis of the third cranial nerve or the ciliary ganglion; or may be due to both causes. It occurs in children, and on taking certain drugs (mydriatics). It occurs also from irritation of the cervical sympathetic <i>directly</i> by incipient lesions in the cervical enlargement of the spinal cord and its mem- branes, or by tumors in the neck, or by excess of car- bonic acid in the blood as in dyspnoea; and <i>indirectly</i> by strong emotions and especially by pain; also in par- alysis of the sphincter pupillae (iridoplegia) from le- sions, such as optic atrophy, glaucoma, lesions of the third nerve, or ciliary ganglion, which break, or im- pair, the reflex arc, and which usually cause more or less diminution of vision and a deficient perception of light; also in coma, in cases of increased intra-cranial pressure, and in some other cerebral and meningeal lesions, especially in their later stages.
	337 Westphal's pupil reaction	When patient's eye- lids are held forc- ibly apart and he attempts to close them he not only turns the eyeball upwards (Bell's phenomenon) but also the pupil con- tracts.	Myosis may be irritative or spasmodic, due to irritation of the third nerve or ciliary ganglion; or may be para- lytic, due to paralysis of the cervical sympathetic gang- lion or nerve, or may be due to both causes. It occurs in old age, in deep sleep, or on taking certain drugs (myotics); also from irritation of the third nucleus or nerve, as in meningitis in early stages and especially in hemorrhage into the pons; and from excessive use of accommodation, as in watchmakers, etc.; also from paralysis of the sympathetic in lesions of the neck and of the spinal cord (syringomyelia). It occurs often in tabes, paresis, iritis, irritation of cornea and, tempor- arily, after excision of the Gasserian ganglion.
	338 Paradoxical pupillary reflex	Pupil dilates instead of contracting upon exposure to light or upon efforts of ac- commodation.	Anisocoria occurs in many conditions and is of little or no diagnostic value.
	339 Mydriasis	Dilated pupils.	
	340 Myosis	Contracted pupils.	
	341 Unequal pupils or anisocoria	One pupil is larger than the other when the eyes are at rest.	

The methods of eliciting the pupillary reflexes are described in Chart I b.
Diseases in which these reflexes are altered are discussed in Chart XIV.

CHART VI

Disorders of Sensation

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the Symptoms of Disease.

344 DISORDERS OF SENSATION

The power of receiving perceptions of the external world and of the occurrences in our own body (the basis of all knowledge) is acquired early in life. The nature of the process is entirely unknown, but it rests upon the power of storing up memories and of recalling them at will. It depends upon the integrity of the central and peripheral sensory neurons (463-4), as well as upon that of the terminal sensory organs and of the cerebral cortex (47 to 56). This power may be diminished, or exaggerated, or perverted in various diseases.

345 DIMINUTION

Either no perception or an abnormally feeble one follows a sensory irritation adequate in health to cause a perception (805, 810).

346 EXAGGERATION

An unusually strong perception, as compared with health, follows any sensory irritation (806).

347 PERVERSION

The occurrence or modification of a perception such as never occurs in health (930).

The conditions under which sensation may be diminished or increased are set forth in Chart VI a.

The conditions under which sensation is perverted are set forth in Chart VI b.

CHART VI a
Diminution and Exaggeration of Sensation
Comprising Numbers 345 and 346, and 348 to 372

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

SENSATION

345 D I M I N U T I O N	DIAGNOSTIC SYMPTOMS	DEFINITION	SIGNIFICANCE
	348 Anesthesia (complete) or Hypesthesia (partial). (Superficial sensibility)	A loss, or diminution, of the normal sensibility to touch upon adequate irritation. Normal sensibility varies in acuteness in different parts of the body and in different individuals.	Diminution of sensibility may be due to disease of the terminal end organs, or to a destructive lesion either of the peripheral sensory neurons (464), (in which case all forms of sensibility are abolished over an area usually coinciding with, but smaller than, the distribution of a peripheral nerve, and the reflex acts in the same part are also abolished); or of the sensory central neurons (463), (in which case frequently all forms of sensibility are not abolished, and the anesthetic area does not correspond to the area of distribution of a nerve, and the reflex acts in the part are not abolished). Sensibility is abolished in coma, narcosis and often apparently in hysteria. A broad zone of analgesia and, more rarely, of anesthesia also, about the body occurs in locomotor ataxia: "tabetic cuirass."
	349 Analgesia or Hypalgesia	A loss, or diminution, of the normal sensibility to pain, which in health varies in different individuals and in different parts of the body.	The anesthetic area may coincide with the distribution of a peripheral nerve or with that of a nerve root (peripheral lesion); or with the distribution of several nerve roots (spinal lesion); or the area may involve one-half the body: called hemianesthesia (cerebral lesion and hysteria). Anesthesia of one side of the face and of the opposite arm and leg, "crossed hemianesthesia," occurs in lesions in the tegmentum of the pons. Anesthesia may involve some portion of the body supplied by small branches of many different nerves, such as a hand, a foot, a leg, a forearm, etc., and be sharply limited "stocking and glove variety" (hysterical). (Fig. 33.)
	350 Thermic Anesthesia or Hypesthesia	A loss, or diminution, of the sensibility to variations in temperature. This loss may be more marked for cold than for heat and vice versa.	
	351 Loss of pressure sense	Inability to distinguish differences in the amount of pressure made on the skin.	
	352 Loss of muscle and joint sense or Akinesthesia. (Deep sensibility)	Inability to tell how strongly a muscle is contracted, whether a joint is flexed or extended, or where an extremity is situated in space. A very complex sensation.	
	353 Apallesthesia or loss of osseous sense or vibration sense.	Inability to feel the vibration of a tuning fork pressed firmly on the skin.	
	354 Astereognosis	Inability to recognize objects by the sense of touch; anesthesia not being present.	Analgesia, thermic anesthesia and apallesthesia may be due to lesion of the central gray matter, or of the antero-lateral ascending tract, of the cord. (Fig. 26.)
	355 Deafness or Anakusia or Hypakusia	Loss, or diminution, of sense of hearing.	Astereognosis always indicates a lesion of the cerebral cortex. (Fig. 15.)
	356 Anosmia or Hyposmia	Loss, or diminution, of sense of smell.	Anakusia, anosmia, ageusia and blindness, may be due to a lesion of the sensory terminal organ, of the sensory nerve or tract, or may be functional. But these symptoms may occur in so many conditions unconnected with the nervous system that they may have very little diagnostic value in nervous diseases.
	357 Ageusia or Hypogeusia	Loss, or diminution, of sense of taste.	
	358 Blindness or Anopsia or Amaurosis	Loss of vision.	Hemeralopia associated with a central scotoma for green and red is not uncommon in tobacco smokers; so that when the pupil is dilated in a dim light the healthy part of the retina can act. This condition is quite different from snow-blindness, where the retina is exhausted by too bright and too long continued light.
	359 Amblyopia	Decided impairment, but not complete loss, of vision, especially for colors in the early stages. Usually in such cases the field of vision is made small by the loss of more or less of its periphery or by scotomata.	Nyctalopia is at times associated with congenital retinitis pigmentosa, with cortical (peripheral) cataract and with other defects in the eye.

	DIAGNOSTIC SYMPTOMS	DEFINITION	SIGNIFICANCE
D I M I N U T I O N (C o n t i n u e d)	360 Hemeralopia	A condition in which the patient sees better in a dim light than in a bright one (day blindness).	Homonymous hemianopia is due to a lesion of the optic tract posterior to the chiasm, of the geniculate bodies, the optic fasciculus or the median surface of the occipital lobe of the opposite side of the brain (lips of calcarine fissure). (Fig. 16)
	361 Nyctalopia	A condition in which the patient sees well in a bright light but is almost blind in a dim one (night blindness).	Bi-temporal hemianopia is due to a lesion of the central part of the optic chiasm. Nasal hemianopia is due to a lesion of the lateral margin of the optic chiasm. Bi-nasal hemianopia cannot result from one lesion.
	362 Hemianopia	Loss of one-half of the field of vision.	
	Homonymous	Loss of the same half in both fields.	Tetartanopia is due to a lesion of the upper lip of the contralateral calcarine fissure if it be a lower quadrant of the field of vision and of the lower lip of this fissure if it be an upper quadrant; very rarely to a partial lesion of the geniculate bodies or optic fasciculus. (Fig. 16.)
	Nasal	Loss of the nasal half in each or either field.	
	Bi-temporal	Loss of the temporal half in both fields.	
	363 Tetartanopia or Quadrantic Hemianopia	Loss of an homonymous quadrant of both fields of vision.	
	364 Achromatopsia or color blindness. Hemichromatopsia	Inability to distinguish the different colors from each other either throughout the whole, or in one-half the field of vision.	Achromatopsia may be due to a congenital defect or to defective education or may be the early stage of a gradually developing blindness or amblyopia. Due to mild, not completely paralysing, lesions of any portion of the visual tract in the broad sense.
	365 Dissociation of sensation	Loss of some forms of cutaneous sensibility (usually for pain and temperature) with preservation of others (tactile). (Figs. 24-7.)	Dissociation of sensation always indicates a lesion of the central gray matter (syringomyelia) or of the lateral columns of the spinal cord, or more rarely a lesion in the ponto-cerebellar angle of the pons at the level of the auditory nerve. It occurs associated with motor paralysis of the opposite side of the body in some cases of Brown-Séquard's paralysis.
	366 Hyperesthesia	Increased tactile sensitiveness. An unusually slight touch can be perceived. A very rare and even doubtful condition. It is usually employed when a touch causes an unusually great, even painful sensation, where hyperalgesia or haphalgesia (380) would be a better term.	
346 E X A G G E R A T I O N	367 Hyperalgesia	Increased sensitiveness to pain.	Exaggeration of sensibility of all kinds is usually functional. More rarely it is the result of an irritative, rather than a destructive, lesion of the central or peripheral sensory neurons. It occurs in strychnine poisoning and tetanus. Hyperesthesia occurs as a zone at the upper limit of the anesthesia in many spinal lesions, and on the same side of the body as is the lesion in Brown-Séquard's paralysis. It is usually associated with increased reflex activity.
	368 Thermic Hyperesthesia or Hyperalgesia	Increased, even painful, sensitiveness to heat or cold, or both.	
	369 Hyperosmia	Increased, even painful, sensitiveness to odors.	Photophobia is functional, or due to eye strain, or to inflammation of some part of the eye, or optic nerve, or cerebral meninges.
	370 Hypergeusia	Increased and unpleasant sensitiveness to taste.	
	371 Photophobia	Increased and painful sensitiveness to light.	Hyperakusia is functional, or due to ear diseases affecting the labyrinth, or to cerebral conditions causing hyperemia of the labyrinth (meningitis, encephalitis, tumors, etc.) and to spinal affections.
	372 Hyperakusia	Increased, even painful, sensitiveness to sounds.	

Methods for the detection of these conditions are described in Chart Ic.

Diseases in which these conditions occur are discussed in Chart XIV.

CHART VI b
Perversions of Sensation
Comprising Numbers 347 and 374 to 392

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

SENSATION

DIAGNOSTIC SYMPTOMS	DEFINITION	SIGNIFICANCE
374 Pain (Figs. 33, 38)	Is an unpleasant sensation not felt in perfect health, except in cases of injury. It varies greatly in intensity. It presents different qualities, such as: tearing, cutting, burning, throbbing, darting, etc. It may be diffuse, or felt in a small area (localized), or may run along a nerve trunk (radiating), or may run half way or entirely about the body or an extremity (girdle), or it may be felt in an area which is itself anesthetic (anesthesia dolorosa). Pains may vary as to time of occurrence, some showing a distinct periodicity (malaria, neuralgia and migraine), some occur at menstrual epochs. Some headaches occur in morning (uremic), others in afternoon (ocular) and others towards evening and at night (syphilitic). Some pains are increased by pressure (neuritis and neuralgia) while some are diminished by it (lead colic).	Perversions of sensibility, especially pain and paresthesiae, are often functional and are often due to irritation (pressure, chemical, inflammatory, etc.) of central or peripheral sensory neurons. Radiating and girdle pains are usually due to lesions of the nerve roots. Anesthesia dolorosa is due to a lesion of the central end of a sensory neuron which has been destroyed below this point and therefore can conduct no sensations from below. Although pain may be felt as peripheral it may be of central origin and due to lesions of central neurons within the brain or cord. On the other hand pains due to lesions in the abdominal viscera may be referred to remote parts of the body or the head (referred pains 952).
347 P E R V E R S I O N S	375 Paresthesiae Curious sensations rarely felt in perfect health, usually unpleasant but not severe enough to be called pain. They are numbness, tingling, formication, heat, cold, heaviness, tired feeling, hunger, etc.	Failure of localization may be functional but usually results from lesions of the peripheral sensory neurons (tabes).
	376 Failure of localization When a cutaneous sensation is felt but cannot be localized. (Topoanesthesia)	Allocheiria occurs in hysteria, very rarely in tabes, hemiplegia and sclerosis. Polyesthesia occurs only in tabes and in hysteria.
	377 Allocheiria When an irritation is not felt at the point of contact, but at a corresponding point on the opposite side of body.	Paradoxical sensation has been met with in a number of spinal and cerebral diseases, but is without diagnostic significance.
	378 Double sensation and Polyesthesia Where one contact gives rise to two distinct sensations (double sensation) or more (polyesthesia).	Haphalgnesia occurs in hysteria.
	379 Paradoxical sensation The quality of thermic sensation is reversed, a hot body feels cold and vice versa.	Retardation of conduction of pain occurs only in lesions of peripheral sensory neurons (tabes or multiple neuritis).
	380 Haphalgnesia A slight tactile impression from certain objects, but not from others, is felt as intense pain.	Persistence of sensation occurs in lesions of the peripheral sensory neurons (tabes).
	381 Retardation of conduction of pain The sensation of pain is not felt until an appreciable interval after the time of contact.	Binocular diplopia is due to a weakness of one or more of the external muscles of one eye, or to displacement of one eyeball; so that the image does not fall on identical spots in the two retinae.
	382 Persistence of sensation The sensation continues an unusually long time after the irritation causing it has ceased to act.	

SENSATION (Continued)

P E R V E R S I O N (C o n t i n u e d)	DIAGNOSTIC SYMPTOMS	DEFINITION	SIGNIFICANCE
	383 Binocular Diplopia (816)	Two separate visual perceptions of the same object, the perception from the normal eye (true image) being more distinct than that from the abnormal eye (apparent image).	Monocular diplopia may occur in hysteria, in cases of double pupillary opening, in anomalous refraction (incipient cataract), and irregularities in the cornea.
	384 Monocular Diplopia or Poly- opia (878- 82)	A condition in which objects appear double or multiple, even when looked at with one eye alone.	Metamorphopsia may occur in hysteria, also in astigmatism (refractive) and in displacement of the retinal elements (retinal) which may occur in retinitis, choroiditis, and in detachment, or tumor, of retina.
	385 Metamor- phopsia	A condition in which objects appear distorted.	Micropsia may occur in hysteria, in paralysis of accommodation and, with distortion, when the retinal elements are spread apart (recent choroiditis or retinitis).
	386 Micropsia	A condition in which everything looks much smaller than normal.	Macropsia may occur in hysteria, in spasm of accommodation and, with distortion, when the retinal elements are crowded together (atrophic stage of retinitis and choroiditis).
	387 Macropsia	A condition in which everything looks much larger than normal.	
	388 Tinnitus Aurium	A sound of ringing, roaring, whistling, etc., in ears or head.	
	389 Parakusis	Perversions of hearing, such as hearing tones incorrectly or hearing better when other loud noises are present at the same time, or hearing sounds or words for which there is no external cause (hallucination).	Tinnitus aurium, parakusis, parosmia and parageusia occur in lesions of the terminal organ and in insanity and functional disorders. They may constitute the aura of an epileptic attack.
	390 Parosmia	The perceptions of abnormal odors or of those for which there is no external cause (hallucination).	Vertigo may be functional (hysteria, neurasthenia, traumatic neuroses); or may depend on changes in the cerebral circulation, especially anemia and hyperemia (cardiac and arterial diseases, congestion in portal or systemic circulation, galvanism of head or neck), or toxic (tobacco, morphine, alcohol, some digestive disturbances, etc.); or may depend on diseases of the cerebellum and its tracts, or of the ear or eye. It is the principal symptom in Ménière's disease (aural vertigo). Vertigo is closely associated with vomiting. In vertigo associated with lesions in, or pressing upon, a cerebellar hemisphere, external objects seem to whirl in the direction away from the injured hemisphere in both conditions, but the subjective vertigo, usually, is away from the injured hemisphere in case the lesion is within it and towards it when the lesion is external and presses upon the hemisphere.
	391 Parageusia	The perception of abnormal tastes or of those for which there is no external cause (hallucination).	
	392 Vertigo	A feeling as if the person (subjective) or as if surrounding objects (objective) were whirling about, or both.	

Diseases in which these conditions occur are discussed in Charts XIV and XV.

CHART VII a
Electrical Examination
Comprising Numbers 395 to 405

CHART VII a
Electrical Examination
Comprising Numbers 395 to 405

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the Symptoms of Disease

	NAME OF THE REACTION	TISSUE TESTED	REACTION TO FARADISM	REACTION TO GALVANISM AND FORMULA OF GALVANIC REACTION	CHARACTER OF THE CONTRACTION	SIGNIFICANCE OF THE REACTION
395 ELECTRICAL REACTION OF MUSCLES AND NERVES (70-3) Nerve fibers respond to changes in intensity of both the faradic and the galvanic currents. The changes in intensity are best brought about by making and breaking the current. Muscle fibers respond only to the galvanic current. The muscle responds to the faradic current only in virtue of the nerve fibers supplied to it. When these nerve fibers are degenerated the muscles can no longer respond to the faradic current. Both nerves and muscles have points on the body surface; the so-called motor points (see figures 1 to 5) from which they are most readily excitable. Therefore, in testing a nerve or muscle by electricity the electrode (positive or negative) is placed on the corresponding motor point (70-3).	396 Normal excitability (473)	NERVE AND MUSCLE	Contraction present to a strength of current which is normal for the nerve and muscle tested.	Neg.Cl.C. Pos.Cl.C. Pos.Op.C. Neg.Cl.Tet. is the normal formula, or in other words Neg.Cl.C. occurs with the weakest current that will cause any contraction. Neg.Cl.C. Pos.Cl.C. with a little stronger current. Neg.Cl.C. Pos.Cl.C. Pos.Op.C. with a still stronger current. The explanation of the above formula is as follows: The weakest current that will cause any contraction of the muscle will do so when the negative electrode is on the motor point and the current is closed. (Neg.-Cl.C.) A more powerful contraction will take place when a stronger current is used and then there will also be a contraction when the current is closed and the positive pole is on the motor point (Pos.Cl.C.). A still more powerful current causes a contraction when the current is opened and the positive electrode is on the motor point (Pos.Op.C.). With such powerful currents and the negative pole on the motor point there results a tetanus or continuous contraction when the current is closed, (Neg.Cl.Tet.); so that the muscle cannot relax to contract again when the current is opened. There is, therefore, in health no reaction corresponding to "Neg.Op.C."	Quick.	Normal excitability shows a normal condition of muscle and nerve.
	397 Diminished excitability		Contraction present but it requires an unusually strong current to produce it.			Diminished excitability occurs in many diseases and conditions, especially in lesions of the central motor neurons and is not of much value in diagnosis.
	398 Exaggerated excitability		Contraction present to an unusually weak current.			Exaggerated excitability is a rare condition. It occurs in nervous persons with moist skins and in tetany.
	399 Reaction of degeneration (472)	NERVE AND MUSCLE	Gradual loss of excitability which becomes complete in about two weeks after injury or onset of the disease. Gradual loss of excitability which becomes complete in less than two weeks after injury or onset of the disease.	No reaction. After the first two weeks the muscle responds to unusually feeble galvanic currents and the normal formula is reversed; the positive pole being more potent. Pos.Cl.C. Neg.Cl.C. Pos.Op.C. Neg.Op.C. (which last reaction never occurs in health). It is usual to express the formula for the normal reaction and for the reaction of degeneration in the German language in which Kathode means the negative electrode and Anode means the positive electrode. The usual normal formula is K.C.C., A.C.C., A.O.C., K.C.Te. The reaction of degeneration is A.C.C., K.C.C., A.O.C., K.O.C. The essence of the normal formula is K.C.C.>A.C.C. The essence of the formula of the reaction of degeneration is A.C.C.>K.C.C.	None. Sluggish. The sluggish character of the muscular contraction is the most characteristic thing in the reaction of degeneration.	The reaction of degeneration proves that the peripheral motor neurons are degenerated and that recovery will either never take place, or will be very slow. The lesion must be either in the peripheral nerves, or nerve roots, or in the anterior horns of the spinal cord, or in the motor nuclei in the brain stem.

ELECTRICAL REACTIONS (Continued)

	NAME OF THE REACTION	TISSUE TESTED	REACTION TO FARADISM	REACTION TO GALVANISM	FORMULA OF GALVANIC REACTION	CHARACTER OF THE CONTRAC- TION	SIGNIFICANCE OF THE REACTION
395 E L E C T R I C A L R E A C T I O N O F M U S C L E S A N D N E R V E S (C o n)	400 Partial reaction of degen- eration	Nerve	Contractions present, but require unusually strong currents, whether faradic or galvanic.	Contractions present to unusually weak currents	Either the normal formula, or the formula of the reaction of degeneration, or a combination of the two may be present. A.C.C. may equal K.C.C.	Quick or sluggish	The significance of this reaction is the same as that of the reaction of degeneration, except that it indicates the lesion is less severe and that all the nerve fibers are not degenerated.
		Muscle	Contractions present only to unusually strong currents.				
	401 Myas- thenic reaction (553)	Nerve and Muscle	Contractions quickly grow less strong and soon cease under rapidly repeated excitation.	Normal	Normal.	Quick, grows rapidly weaker and ceases.	Occurs only in myas-thenia gravis (553).
	402 Myo- tonic reaction (613)	Nerve and Muscle	Continuous tonic contraction lasting some time after the electrical stimulation has ceased.	Curious wave-like contractions occur, and last after electrical stimulation has ceased.	Positive pole is about equally as potent as the negative. Hence the formula A.C.C. = K.C.C.	Continues usually a long time and has a wave-like character.	Occurs in Thomsen's disease (613).
403 Neuro- tonic reaction		Nerve	Unusually excitable. Tetanic contraction persists after electrical stimulation has ceased.		Normal.	Continuous	Occurs in hysteria, amyotrophic lateral sclerosis and chronic bulbar paralysis.
		Muscle	Normal.	Normal.			
404 Reaction of com- pletely degenerated muscle (70 to 73)		Muscle	None.	None.	None.	None.	Muscle fibers are entirely degenerated and recovery is impossible.
405 Electrical reaction of the Optic and Auditory Nerves	<p>The optic nerve responds to the galvanic current with a sensation of light, the color of which varies with the pole employed.</p> <p>The auditory nerve responds with a loud sound when the negative electrode is placed in or near the meatus and the current closed and with a faint sound when the positive pole is used and a stronger current broken. These reactions are without diagnostic importance.</p> <p>The negative electrode placed in front of the ear causes a nystagmus towards the ear tested when the current is closed and in the opposite direction when the current is broken. The positive electrode causes nystagmus in exactly the reverse direction.</p>						

In cases of disease in which the caloric test (79) is absent and in which the electric test is present, it is fairly certain that the lesion is in the labyrinth and not in the nerve. If there is no response to either the caloric or the electric test the lesion is in the nerve or its nucleus.

CHART VII b

Erb's Motor Points for Electrical Examination of Nerves and Muscles

The motor points are the areas upon the surface of the body at which the individual nerves and muscles can be most easily excited by electricity. For the nerves, these points coincide with those at which the nerve lies most superficially or where it can be pressed against a resisting tissue; for the muscles, they lie over the point of entrance of the nerve into the muscle.

Comprising Figures 1 to 5

ERB'S MOTOR POINTS

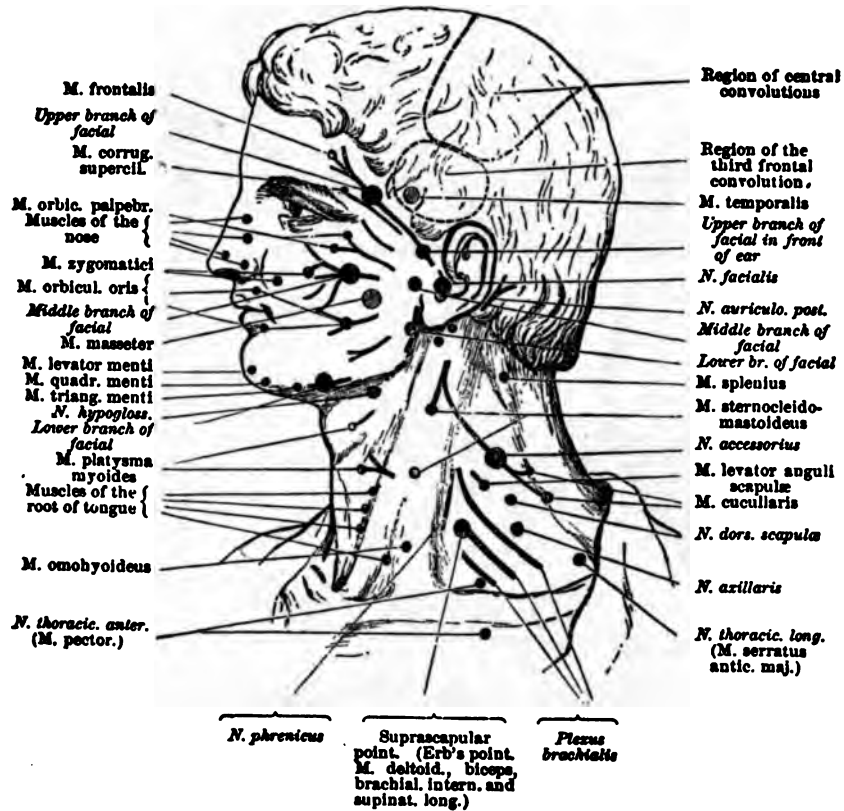


Fig. 1

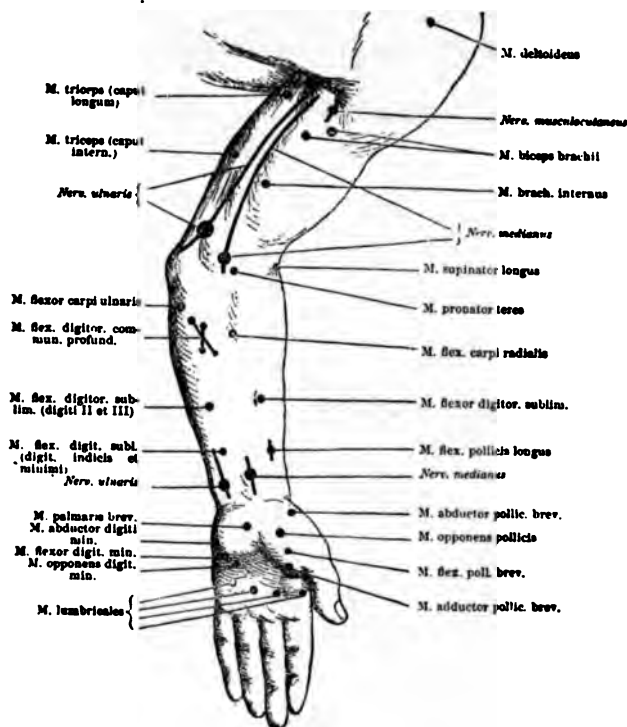


Fig. 2

ERB'S MOTOR POINTS (Continued)

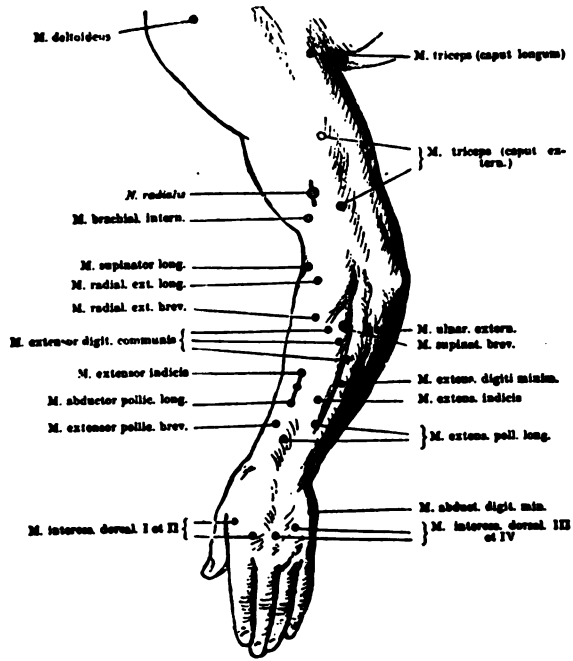


Fig. 3

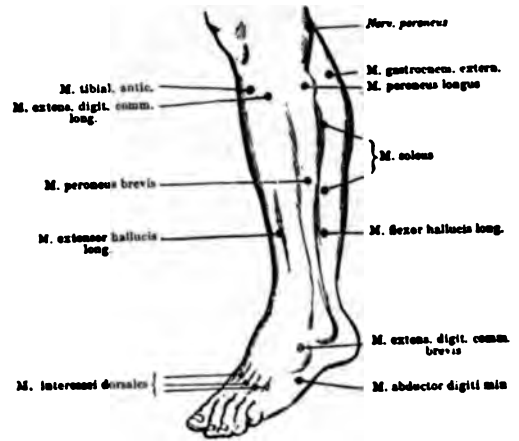
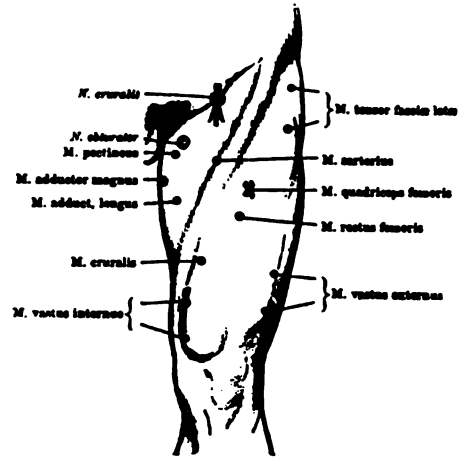


Fig. 4

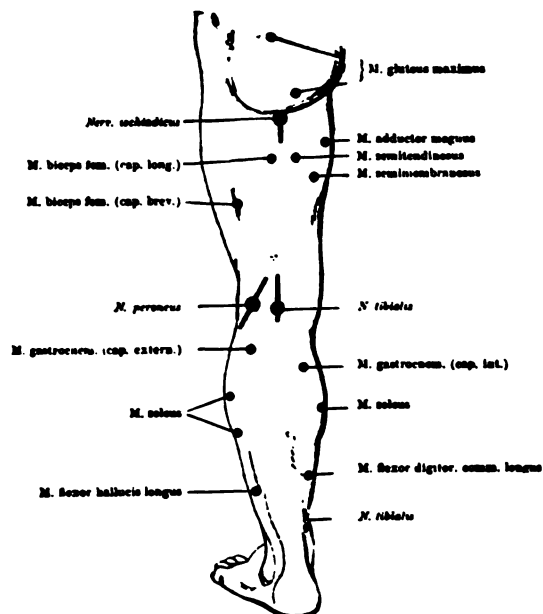


Fig. 5

CHART VII c

ERB'S DIAGRAM SHOWING THE EFFECTS OF INJURY OF A NERVE

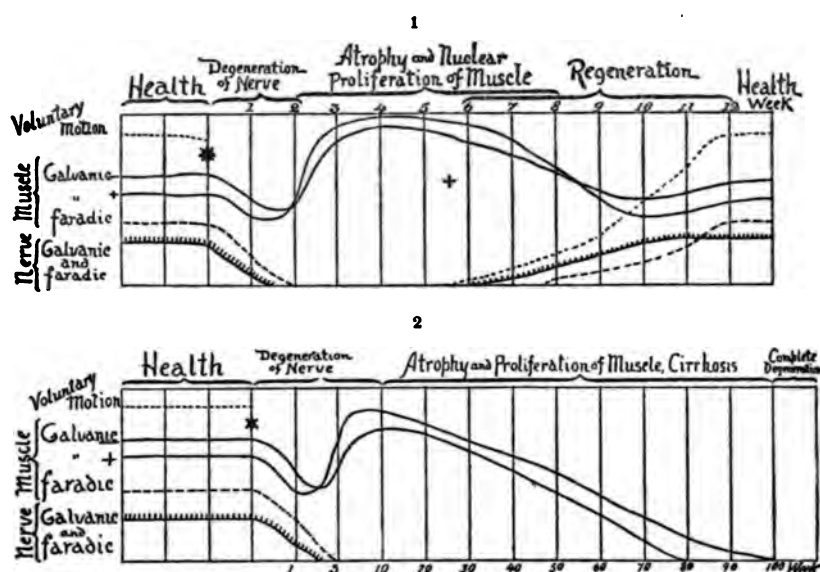


FIG. 6

Charts Illustrating the Reaction of Degeneration

The star (*) indicates the incidence of a paralyzing lesion in the domain of the peripheral neuron. Voluntary motion is lost at once. During the first two weeks there is slight diminution of the galvanic excitability of muscle; there is also rapid diminution of the faradic excitability of muscle and of the galvanic and faradic excitability of nerve, which are completely lost at the end of the second or third week. During the second week there is rapid increase in galvanic excitability of muscle and the response to the positive pole becomes greater than to the negative.

Chart 1 represents the reaction in a case terminating in recovery. During the sixth week (indicated by the cross X) regeneration begins. The increased galvanic excitability of the muscles gradually diminishes until it becomes normal and the poles are reversed so that the negative response is again greater than the positive. Voluntary motion returns first, then the galvanic and faradic excitability of the nerve, and last of all, the faradic excitability of the muscles.

Chart 2 represents the reaction in a case terminating in atrophy and cirrhosis of the muscle. The galvanic excitability of the muscle is increased and the poles are reversed, as before. The decline in galvanic excitability continues, however, until the end of the second year, when it is entirely lost. Voluntary motion, and the electrical reactions of both muscles and nerve are thus permanently destroyed.

CHART VIII
Analysis of the Cerebro-Spinal Fluid
Comprising Numbers 410 to 420

ANALYSIS OF THE OBJECTIVE SYMPTOMS OF THE CASE (SEMEIOLOGY)

ABNORMAL CEREBRO-SPINAL FLUID

CHARACTER- ISTICS	METHOD OF TESTING	SIGNIFICANCE
411 Tension	Can be roughly estimated by the rapidity of flow of fluid through the canula, whether in drops or a stream, more accurately by the height to which the fluid rises in a vertically held glass tube connected by a short rubber tube with the canula. A stopcock on the canula adds to the accuracy by preventing the escape of much fluid and the consequent lowering of the tension. The fluid in the tube rises and falls with the respiration. An additional more rapid and stronger pulsation indicates a basilar aneurism.	A low or very rapidly diminishing tension has no diagnostic meaning, except as indicating an obstruction to the communication of the fluid in the ventricles with that of the vertebral canal, as in closure of the foramen of Magendie. A high tension means increased intra-cranial, or intra-spinal, pressure caused by an increased amount of cerebro-spinal fluid or by a foreign body within the cerebro-spinal cavity. It occurs in tumors, abscess, hydrocephalus, hemorrhage, acute, sub-acute, some cases of chronic, and serous meningitis, also in cerebral edema (nephritis, anemia, etc.), acute infectious diseases and some other conditions.
412 Red color or 413 reddish yellow color	By sight. Hematoidin crystals may be seen under the microscope.	Fresh blood in the fluid may be the result of puncture of a blood vessel, in which case it is most abundant in the fluid first drawn, usually coagulates, and settles quickly on centrifugalization. Or, May be the result of hemorrhage into the ventricles or membranes. Hemorrhage, haematoma, aneurism, etc.
414 Cloudy	By sight. Pus cells under the microscope. Polymorpho-nuclear leucocytes.	An increase of cellular elements in the fluid is usually the result of an acute or sub-acute meningitis. In some cases of acute meningitis, however, the fluid may be clear.
410 A B N O R M A L C E R E B R O S P I N A L	415 Clear with delicate coagulum By sight. 416 Cellular elements and bacteria Fluid soon after withdrawal should be centrifugalized. Tube should be emptied quickly and from its walls and bottom sediment should be sucked in and out of a capillary tube, well mixed and spread on two clean slides. One slide should be stained by Gram's method for bacteria, and the other by Wright's blood stain for cellular elements. Or, The fluid (not centrifugalized), 10 parts, can be mixed with 1 part of a solution consisting of methylene blue 0.2%, glacial acetic 4.0%, and water to 100 %, and counted in a Thoma-Zeiss chamber.	Tuberculous meningitis, usually. The normal cerebro-spinal fluid shows under these conditions 1 to 3 cells in a field of the microscope. If there are more than 4 to 6 cells in a field it indicates a meningitis. If the cells are mainly leucocytes it indicates epidemic cerebro-spinal, or purulent meningitis, or rarely an acute tuberculous meningitis. Broadly speaking, an acute infectious meningitis. If the cells are mainly or entirely lymphocytes it indicates a tuberculous meningitis, or cerebro-spinal syphilis, or paresis, or tabes, or acute anterior poliomyelitis, or convalescence from any form of acute meningitis. Broadly speaking a chronic infectious meningitis. If echinococcus cysts or hooklets are present, they indicate the presence of an echinococcus cyst.

ABNORMAL CEREBRO-SPINAL FLUID (Continued)			
F L U I D	CHARACTER- ISTICS	METHOD OF TESTING	SIGNIFICANCE
	417 Sugar	By Haines' test or other tests.	Not of much significance, but the sugar normally present is diminished usually in meningitis and in some other conditions.
	418 Albumen	Two c.c. of the fluid mixed with 10 c.c. of Esbach's fluid is centrifugalized during one hour in a conical tube graduated to 0.1%.	Normally not more than $\frac{1}{4}$ % is present. Usually increased in meningitis and tumors. A diminution in the amount usually indicates a progressive space-occupying disease. Of little diagnostic significance.
	419 Globulin	Two c.c. of a saturated solution of chemically pure neutral ammonium sulphate should be placed in a test tube and one cc. of the cerebro-spinal fluid should be gently run upon its top. If the reaction is positive, within 3 minutes, a grayish white ring should form at the junction of the two fluids. At the end of one-half hour, the surface of the ring should show a delicate network. Best seen by indirect illumination. Or, Boil slightly 1 volume of the cerebro-spinal fluid with 5 volumes of a 10% butyric acid solution, add 1 volume of a normal solution of sodium hydroxide, reheat and allow to cool. If a flocculent precipitate forms, the reaction is positive. (Noguchi test).	Indicates meningitis, acute anterior poliomyelitis, cerebro-spinal syphilis, paresis, tabes, rarely a brain tumor.
	420 Positive Wasser- mann reaction	This test can only be performed in a laboratory by an expert.	The reaction is positive in 90% of cases of paresis and in 60% of cases in tabes. In cerebro-spinal syphilis both the cerebro-spinal fluid and the blood usually give a positive reaction. In other cases of syphilis (without meningitis) the reaction is usually negative with the cerebro-spinal fluid, but positive with the blood.

CHART IX
Special Syndromes and Anatomical Terms
Comprising Numbers 425 to 465

SYNDROMES AND SPECIAL SYMPTOMS OF DISEASE

SYNDROME	DEFINITION	SIGNIFI- CANCE
425 Hysterical symp- toms (1074)	Occur usually in self-conscious females of an emotional nature. Lack of inhibition and great susceptibility to suggestion. Desire to excite admiration and sympathy and wonder. Mental instability. Globus hystericus (426). Spinal, inguinal (or ovarian) and other tenderness. Great variety of symptoms (especially subjective) which cannot be explained by any organic lesion. Glove and stocking form of anesthesia or hemianesthesia and concentric contraction of the field of vision are common symptoms, but the patient is usually ignorant of their existence until they are discovered, or more probably suggested, by the physician. Exaggerated reflexes but no ankle-clonus or only pseudo-clonus. Never a Babinski reflex. Motor paralyses, tremors, contractions and convulsions are not uncommon. Transference of hemianesthesia can be effected in some cases. The anesthesia of the fingers does not prevent delicate acts being done by them with eyes closed. Such patients when tested and the anesthetic area is touched often answer "no" at the moment of contact (48). Many other symptoms do not seem to be real but rather seem to be imaginary and may result from hallucinations or delusions or more probably are the result of suggestion: auto-, or foreign. Probably many of the hysterical symptoms come into existence as the result of the physician's careful and minute examination or repeated examinations (foreign suggestion).	Hys- teria (1074)
426 Globus Hystericus (1074)	The feeling of a lump or ball behind the upper end of the sternum which interferes both with swallowing and breathing. The sensation often commences in the epigastrium and rises to the base of the neck and remains there; the patient not being able to get it up or down. It may be caused by a spasmodic contraction of the muscles of oesophagus or throat.	
427 Hystero- genic areas (1074)	Spots scattered over the body, but usually in the left inguinal region, where light pressure or irritation will cause more or less violent hysterical attacks	
428 Hystero- frenic areas (1074)	Spots scattered over the body, but usually in the left inguinal region, where firm and continued pressure will cause the arrest of an existing hysterical attack.	
429 Lasègue's symp- tom (1074)	A condition in which the patient cannot move an anesthetic extremity when her eyes are closed, but can move it readily when she opens her eyes and looks at it.	
430 The epi- leptic aura (575, 846, 1058)	The aura is a symptom (warning) which occurs before the attack in about half the cases of epilepsy. It may be remote or immediate. The former is often called "a prodromal symptom" and occurs hours and days before the attack. It consists usually in an emotional change (irascibility, etc.), changes in the amount of sleep, of food taken, in sexual desire and vaso-motor phenomena. Much more characteristic and important is the immediate aura which occurs a fraction of a minute before the attack. This aura may be "psychic" (anxiety, anger, joy, dreamy states, special thought or memory, etc.), or a "sensory hallucination" which may be visual (blindness, lights, colors (red), elaborate false visual perceptions, etc.), or auditory (deafness, noises, and false auditory perceptions), or olfactory or gustatory hallucinations, or cutaneous paresthesiae (the feeling of a wind blowing on some part of the body is quite common) and pains, or visceral paresthesiae, especially epigastric. Vertigo is a common immediate aura; or the immediate aura may be motor and consist in twitching of a group of muscles, (Jacksonian epilepsy), or in more complicated automatic movements of the body, or in hiccough, sneezing, yawning or swallowing. Vaso-motor disturbances, flushing or pallor with secondary paresthesiae, are not uncommon immediate auras. Usually the aura is always the same in the same individual; rarely it varies. In rare cases the aura may not be followed by an attack and in still rarer and always doubtful cases it may be the only symptom of epilepsy.	Epi- lepsy (575, 846, 1058)

SYNDROMES AND SPECIAL SYMPTOMS OF DISEASE (Continued)

SYNDROME	DEFINITION	SIGNIFICANCE
431 Jacksonian epilepsy (587-8, 605)	A clonic spasm of one or more muscles in one side of the face or in one arm or leg, which may remain local, but usually rather rapidly extends to other muscles of the same side of face, or of the arm or leg in which it commenced. It then may extend to an adjacent extremity in the same order in which the cortical centers are placed: thus from the face to the arm and then to the leg, from the leg to the arm and then to the face, from the arm to the leg and face nearly or quite simultaneously, but never from the face to the leg, or vice versa, without involving the arm. When the spasm has extended over the whole half of the body it may remain so or may pass across and involve the other side. As long as the spasm is local or limited to one-half of the body consciousness may or may not be lost, but when the spasm involves both sides of the body consciousness is always lost.	Local cortical lesion (587-8, 605) (Figs. 15-16)
432 The prodromata of apoplexy (504, 1060-3)	In many cases of apoplexy, especially in cases of cerebral thrombosis, the apoplectic attack is preceded by a number of more or less definite and characteristic symptoms which may be remote, preceding the attack by months or years; or immediate, occurring immediately before the attack. These prodromata are both <i>general</i> , such as headache, vertigo, drowsiness and stupor, irritability, forgetfulness, hypochondriacal feelings, ringing in the ears, flashes before the eyes, etc.; and <i>local</i> , such as temporary attacks of aphasia, diplopia, achromatopsia, dysarthria, temporary paralysis of arm or paresthesiae. None of these symptoms is so characteristic that an attack of apoplexy can be confidently predicted from its presence. The most constant prodromal symptom of apoplexy (except of embolism) is high arterial tension.	Apoplexy (504, 1060-3)
433 Tabetic or visceral crises (661)	Paroxysmal attacks of pain in, and functional disturbances of, some viscera, occurring in the course of locomotor ataxia. These attacks recur after irregular intervals, persist during an hour, or a day or two, and are analogous to the paroxysmally occurring lightning-like pains in the legs. "Gastric crises" are the most frequent and consist in severe pain in the epigastrium together with uncontrollable vomiting and retching. At times attacks of gastric pain or of vomiting occur separately. "Hepatic crises" resemble gallstone colics, even being accompanied by slight jaundice at times. "Laryngeal crises" consist in attacks of coughing and dyspnoea. "Laryngeal vertigo" (Ictus laryngeus) consists in a sensation of tickling and burning in the larynx, a stridulous inspiration with a feeling of suffocation and a falling to the ground unconscious for a few minutes. "Pharyngeal crises" consist in repeated acts of noisy swallowing. "Renal crises" resemble attacks of renal colic. "Vesical crises" consist in pain in region of bladder and prostate, and constant desire to urinate. "Urethral crises" consist in attacks of pain in urethra and desire to urinate. "Rectal crises" consist in attacks of pain in the rectum and tenesmus. "Vulvo-vaginal crises" consist of attacks of pain in vagina. "Clitoridian crises" consist of attacks of pain in vulva with sexual desire and discharge of mucus. "Anginal crises" resemble angina pectoris. Occasionally "crises" of several kinds occur simultaneously.	Tabes (661) (Fig. 27)
434 Bulbar symptoms (546)	A combination of several or all of the following symptoms, dysarthria or anarthria (283-4), dysphagia (285), drooling of saliva from mouth, propulsive speech, and puffing of lips. Paralysis of the 7th, 9th, 10th, 11th, and 12th, and at times of other cranial nerves. Spastic paraplegia or hemiplegia of extremities. Sensory paralysis and ataxia. Respiratory difficulty, and in severe cases rapid, irregular pulse and Cheyne-Stokes' respiration.	Lesion or disorder of medulla (546). (Figs. 21-2)
435 Cheyne-Stokes' respiration	Long pauses in the respiration. After a pause the respiration commences slow and deep and rapidly becomes quick and superficial and as rapidly becomes slow and deep again and terminates in another long pause (lasting from five to sixty seconds, or more) and so on; each cycle being completed in a few minutes. A somewhat similar respiratory disturbance which is called Biot's respiration consists of frequent pauses in the respiratory act, lasting many seconds. Biot's respiration occurs in Bright's disease, etc., but has no particular significance in nervous diagnosis.	

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PART II

DIFFERENTIAL DIAGNOSIS

A CLINICAL DIAGNOSTIC ANALYSIS OF THE SYMPTOMS

OBTAINED FROM THE EXAMINATION OF PATIENTS

INTRODUCTION TO THE DIAGNOSTIC CHARTS

DIRECTIONS FOR THEIR USE

In using this book for diagnostic purposes the student, or practitioner, having made a complete examination of the patient according to the scheme presented in chart I, should make note of the more important symptoms. Then, selecting any one of these symptoms, he should turn to the table of contents and see which chart treats of the disorders which include this symptom. Finally, turning to the commencement of the chart indicated, he should apply one test after another until he reaches the diagnosis.

At the left margin of each chart is placed the symptom to be analyzed; on the right margin are placed all the possible diseases in which this symptom can occur. Proceeding from left to right, in each column a number of alternatives are offered, and by selecting the one appropriate to the case the diagnostician proceeds from one column to the next, until he reaches the correct diagnosis. In the column immediately preceding the diagnosis is an abstract of most of the symptoms which may occur at different stages of the disease. The great majority, but not necessarily all, of the symptoms given in the abstract should be present in the history or found in the examination of the case, if the diagnosis be correct.

A few practical examples will illustrate the method much better than a long general description. Let us then consider a few cases as they occur in actual practice. Only the essential symptoms are noted.

Case I. Male, aet. 51.—He smoked and chewed tobacco and drank to excess for years. About two months ago he began to have pains at various points in both legs. His legs became slowly weaker and his flesh became tender, but he is able to walk a mile. Organic reflexes normal. Feet cold, and the legs have lately grown smaller. On physical examination the muscles of the lower legs, and less so those of the thighs are weak, tender and somewhat atrophied. The legs, and especially the feet show slight anesthesia, marked analgesia and well marked retardation of the conduction of pain. Achilles reflex absent. A slight knee-jerk can be obtained with difficulty. In walking toes drop a little and the knees are raised abnormally high.

Important symptoms: PARALYSIS (weakness), HIGH-STEPPING GAIT, ANESTHESIA and PAIN.

The chief symptom in this case is weakness, and we, therefore, turn to chart X, which discusses "diseases causing motor paralysis." The paresis in this case is continuous and the reflex acts are diminished or absent. We, therefore, have to do with a flaccid paralysis and turn to chart Xa. The presence of muscular atrophy following the paralysis, together with the absence of any apparent hypertrophy, guides us in the second column away from the functional diseases and the muscular dystrophies and to the degenerative atrophies; while the normal organic reflexes guide us in the third column away from the spinal cord, and to the peripheral nerve, diseases. The presence of anesthesia, pains, muscle tenderness and other sensory symptoms guide us in the fourth column to the class of neuritis of the spinal nerves. In the fifth column the fact that there are many spinal nerves affected guides us to the diagnosis: Multiple Neuritis, which the history of alcoholic abuse confirms.

We can approach this case in another way by considering his abnormal walk. In the table of contents we find that disorders of gait are treated in chart XIII and indeed in chart XIIIc. The walk in this case is evidently "paralytic and flaccid," the tendon reflexes being diminished. Furthermore it is a high-stepping gait. A comparison of the three possible abstracts with the symptoms of our case makes it evident that the case is one of Multiple Neuritis.

We can trace the case also by means of the anesthesia and analgesia: symptoms which are discussed in chart XIVa. The tendon reflexes being diminished and the organic reflexes normal in this case, we are led to three abstracts, only one of which fits our case, and thus the diagnosis

of Multiple Neuritis is again confirmed. Finally we may take up, the initial symptom in the case: pain in the legs. Pain is discussed in chart XV and pain in the extremities in chart XVc. In our case the pain is bilateral and is associated with anesthesia; so that we are again brought to three abstracts, of which the one of Multiple Neuritis most nearly fits our case.

Case II. Female, aet. 23.—Ten years ago she and her brother had simultaneously an attack of headache, backache and fever. Her brother died and she recovered with a paralysis of both legs, which has since improved, rapidly at first, then more slowly. Her legs are still somewhat weak, especially the left one, but she can walk fairly well. No sensory disturbances, organic reflexes normal. On physical examination there is a decided weakness, slight atrophy and slight shortening of left leg. Knee-jerks are absent in both legs. No objective sensory abnormalities.

Important symptoms: PARALYSIS.

In the analysis of this case we follow the same path traced in case I until we reach column four in chart Xa. In this case there are no sensory symptoms, the paralysis involves neither the cranial nor the extensor nerves exclusively, and is acute in its origin; so that the diagnosis must be Acute Anterior Poliomyelitis.

Case III. Male, aet. 48.—Had a chancre followed by a cutaneous rash twenty-two years ago. During the past ten years has had "lightning pains" in legs and a girdle sensation, also gastric, vesical and urethral crises. During the past six months, his walking has become difficult and awkward and is much worse, practically impossible, in the dark. Organic reflexes normal, except for some delay in micturition. On physical examination there is no loss of muscular power, but all movements of legs are awkward, violent and excessive. There is marked ataxia, anesthesia in areas and well marked retardation of conduction of pain from feet. Complete absence of knee-jerk. Argyll-Robertson's pupillary reflex, Romberg's symptom and loss of muscle sense in legs. Lumbar puncture gave fluid showing the presence of globulin and lymphocytosis and a positive Wassermann. In walking the patient does not stagger, but flings feet out widely.

Important symptoms: ATAXIA, ATAXIC GAIT, ANESTHESIA, PAIN, ABDOMINAL CRISES, and LYMPHOCYTOSIS IN CEREBRO-SPINAL FLUID.

In this case there is no loss of motor-power but well marked ataxia in legs. From the table of contents we learn that disease causing perversion of motion, including ataxia, are treated in chart XIIa, to which we turn. As the patient does not stagger in walking and the movements of the legs are ataxic, not only in walking, but also in other movements, it is certain that the case is one of "motor ataxia." The ataxia is bilateral and the knee-jerks are absent; so that it is evident that we have to do with tabes or multiple neuritis (pseudo-tabes). We differentiate these two diseases by comparing the abstracts of their symptoms. As in this case there is no muscular weakness, atrophy and tenderness, it is plain that the diagnosis is Tabes.

We may also reach a diagnosis in this case by studying the patient's walk with the aid of chart XIIIc. The gait is ataxic, rather incoordinated than staggering, the knee-jerks are abolished and there is Argyll-Robertson's phenomenon, so that the diagnosis of Tabes is confirmed. Furthermore we may trace the case by the symptom of anesthesia with the aid of chart XIVA. The tendon reflexes are abolished. The organic reflexes are not much disordered, but they are slightly. There is no motor paralysis and thus we are led again to Tabes. If we consider the symptom named "Argyll-Robertson's phenomenon," which is present in this case, we shall find it discussed in chart XIVd and here again we are led directly to Tabes. If we consider the pains in the legs or the girdle sensations about the body or the abdominal crises, we find them discussed in chart XV and in either case are led to Tabes. If we consider the results of the examination of the cerebro-spinal fluid with the aid of chart XIX, we find the butyric acid test positive, the existence of lymphocytosis, a positive Wassermann, a clear fluid and ataxia, and thus the diagnosis of Tabes is again confirmed.

Case IV. Female, aet. 19.—Patient's father and mother were first cousins. They had eight children, of whom three died in infancy and four are healthy. Child learned to walk late and with difficulty, frequently stumbled and fell. Was backward at school and when she was nine years old it was evident to all that she was not normal. Patient's movements became gradually and steadily more awkward. Now she cannot walk without aid. General movements are slightly ataxic and simulate somewhat a tremor. Movements of the legs are more ataxic and weaker than those of the arms. Her walk is extremely ataxic and staggering. No knee-jerks, Babinski present. Organic reflexes normal. Internal strabismus. No loss of muscle sense.

Important symptoms: ATAXIA and ATAXIC GAIT.

The most characteristic symptom in this case is ataxia and so, as in case III, we turn to chart XIIa. In this case, the ataxia is mainly on walking and there is no motor paralysis and no loss of muscle sense. We are, therefore, brought to the alternative as to whether the disease occurs in an adult or a child. This case doubtless dates from early childhood. There are no similar cases among her brothers and sisters, but she comes certainly from a tainted family. She has no nystagmus, but has strabismus. As this case began before puberty and has no knee-jerks it is doubtless a case of Friedreich's Ataxia. The strabismus points to Marie's hereditary cerebellar disease and indeed these two diseases are so closely related that there is some question as to whether they are separate entities.

We may approach this case from a different angle. The chief symptom is difficulty in walking. We turn, therefore, to chart XIIc and note that the walking is ataxic. The staggering gait which is permanent, the bad heredity, the absence of knee-jerk and the commencement of the disease in infancy confirms the diagnosis of Friedreich's Ataxia. It may be noted in passing that this case does not show a distinct tremor, or nystagmus, or the blurred speech which symptoms are often present in this disease.

Case V. Male, aet. 62.—His disease commenced with difficulty in speaking and swallowing about a year ago, and has slowly and steadily progressed. His speech has become so bad that it is unintelligible and he has the greatest difficulty in swallowing, and chokes over his food. There is constant drooling of saliva from his mouth. Cannot protrude his tongue beyond his teeth, cannot raise his arms because of weakness of muscles about the shoulders. His legs are somewhat weak. Fibrillary contractions and great atrophy of muscles of tongue and of shoulder girdle (deltoid, pectorals, etc.). Muscles of hands are not involved. Absence of tendon reflexes in arms. Knee-jerks lively, ankle-clonus and Babinski are present. There are no sensory disturbances.

Important symptoms: PARALYSIS, FIBRILLARY CONTRACTION and MUSCULAR ATROPHY.

The principal symptom in this case is a motor paralysis. We turn, therefore, to chart X. The paralysis certainly is a continuous one and of the three alternatives next offered us we must select the third, inasmuch as we have a flaccid paralysis with muscular atrophy in the head and arms and a mild spastic paralysis in the legs. We turn, therefore, to chart Xc. In this case the cranial and spinal nerves are involved, next there are no sensory symptoms, next the disease is chronic, and finally the lips, tongue, larynx and pharynx are involved; consequently the diagnosis is Progressive Bulbar Paralysis. But this diagnosis does not explain the paralysis and atrophy of the muscles of the shoulder which are supplied by spinal nerves. We turn, therefore, to the next sub-division, where spinal nerves are alone involved, and follow through, no sensory symptoms and through a paralysis involving the shoulder girdle muscles, and reach the diagnosis of Amyotrophic Lateral Sclerosis. The diagnosis is, then, a combination of two diseases: Progressive Bulbar Paralysis and Amyotrophic Lateral Sclerosis, and we find in the abstracts of these two diseases that they often occur together in combination.

If we consider the symptom "fibrillary contraction" with the aid of chart XIIb, it is evident that this is an organic and not a functional disease, that there is a marked muscular atrophy and that there are no sensory symptoms, and thus the diagnosis of both Progressive Bulbar Paralysis and Amyotrophic Lateral Sclerosis is confirmed. Finally if we consider the symptom

"muscular atrophy" with the aid of chart XVIIa we find that the atrophy is considerable and of a relatively rapid course, that there are no muscular hypertrophy and no sensory symptoms and thus we are led again to the same diagnosis.

Case VI. Male, aet. 12.—During the first year of his life the child had great difficulty in retaining his food. At the end of his first year he began to have convulsions with unconsciousness, and with the exception of an interval of two years these have continued up to the present time; the last attack having occurred three weeks ago. The child has a very small head and an idiotic expression of face. He apparently understands most of what is said to him, but he can talk only a very little and only a few words are intelligible. There are no contractions or deformities, and he uses his arms and legs well.

Important symptoms: ANARTHRIA and IDIOCY.

The most striking symptom in this case is that a boy of twelve years can scarcely speak intelligently. Turning to the table of contents we find that disorders of speech are treated in chart XIII, to which we turn. The loss of speech in this case is so nearly complete that it can be called anarthria, which is discussed in chart XIIIa. The disease is evidently congenital, and the expression of the face is idiotic, and reading and writing are impossible; so that the diagnosis is Idiocy. Had we on the other hand decided that the child could speak, but very imperfectly and unintelligibly we should have sought for the disease in the same chart XIIIa, under the heading of dysarthria. Here the congenital nature of the defect and the absence of cleft palate, etc., would have led us directly to Imbecility. In order to trace the case further let us follow the cross reference after idiocy which is 1081 and which we find in chart XVIc. This case on account of his convulsions might be classed under Epileptic Idiocy or on account of his small head under Microcephalic Idiocy, or under both.

Case VII. Female, aet. 53.—Complains of trembling and that she cannot execute any movements quickly, because her arms and legs are stiff and rigid. When walking she has a decided tendency to pitch forward. Feels warm at times when the room seems cool to others. Expressionless face, passive tremor of hands. Propulsion and retropulsion when walking or standing. Rigidity of arms and legs. Difficulty in rising from a low chair. Knee-jerks rather increased.

Important symptoms.: MUSCULAR RIGIDITY, TREMOR and ABNORMAL WALK.

The most characteristic symptom in this case is the rigidity of the arms and legs which is a mild tonic spasm. From the table of contents we learn that diseases causing spasm are treated in chart XI, to which we turn and find that general tonic spasm is discussed in chart XIb. In this case there is no fever and of the five sub-divisions under this head, this case clearly falls in the second: "rigidity which does not prevent passive or voluntary motions." Of the two alternatives next offered it is evident that we must choose the second, in the abstract of which we find all the symptoms present in our case. The diagnosis is, therefore, Paralysis Agitans.

If we follow the symptom "tremor," we find this treated in chart XIIb. It is a passive tremor and, whether it be increased or diminished on voluntary movements, if it be slow, the abstracts show that it is a case of Paralysis Agitans, because the other abstracts do not fit this case at all.

If we consider the difficulty in walking in this case we turn to chart XIIIc. In the three great divisions offered this case evidently falls in the third: "paralytic and spastic;" and of the two sub-divisions next offered we must take "general rigidity" which leads us again to the diagnosis of Paralysis Agitans.

Case VIII. Male, aet. 59.—During the past 34 years has had at times attacks of asthma. During the past four years has been troubled by a great many paroxysmal attacks of vertigo, at irregular intervals; some are slight, some are so severe as to throw him from a chair half way across the room to the floor, where he must lie for several hours, because when he raises his head from the floor he vomits violently and the dizziness becomes worse. He often has slight attacks of vertigo, which make him stagger when walking. During these four years he has been slowly

growing deaf in his left ear; the deafness being now extreme. He has also had during the same time in the same ear, a buzzing and a ringing which is most intense just before an attack of vertigo. He has no paralysis and no loss of muscle sense. Bone conduction is absent. He also is much troubled by gastric flatulence, to which he attributes his vertigo, but when he takes digestive medicine and the digestive disturbances are relieved, the vertigo remains unchanged. His eyes were found to be astigmatic and proper glasses used, but no improvement in the attacks of vertigo followed.

Three years after the above record was made the attacks were milder and less frequent, but at that time his left ear was totally deaf and deafness was advancing in his right ear.

Important symptoms: PAROXYSMAL VERTIGO, STAGGERING WALK and DEAFNESS.

In this case the principal symptom is paroxysmal attacks of vertigo; diseases causing which, we learn, are treated in chart XVd. We see from this chart that vertigo may be caused by digestive disturbances and disease of the eye, both of which were present in this case, but the vertigo persisted when these abnormal conditions were relieved; so that they could hardly be the cause. On the other hand, we find that vertigo is associated with deafness: a prominent symptom in this case, and in looking over the abstract of this form of vertigo we see that it fits the case exactly; so that the diagnosis is Ménière's Disease.

If we consider another symptom "the occasional staggering in walking," we find this treated in chart XIIa. This patient has no loss of muscle sense and no muscular paralysis. He is an adult and his hearing is abnormal and thus we are led again to Ménière's Disease. Finally if we trace the symptom "deafness" with the aid of chart XIVE we find that the deafness, at any rate at first, was unilateral, that bone conduction is absent, that there is no facial paralysis and that severe paroxysmal vertigo and tinnitus aurium is present; thus confirming again the diagnosis.

Case IX. Female, aet. 17.—Heredity good. Was well until about three years ago when, at the time of commencing menstruation, she began to have attacks of clonic convulsions with unconsciousness, which have continued up to the present time and in which she has occasionally bitten her tongue. Has also lesser attacks of unconsciousness, or very cloudy consciousness in which she automatically prays, or says foolish things. Has no memory of any of her attacks. She has an immediate aura of fire before her eyes and of wheels revolving in her head. Some headache follows the attack. The convulsions occur only, and the lesser attacks mainly, at night. Physical examination is negative, urine normal. Fundus of eye normal. Knee-jerks equal. Much acne on face.

Important symptoms: COMA and CONVULSIONS.

The constant symptom in all her attacks is unconsciousness, or coma, of short duration. This symptom is treated in chart XVIa. There is no history of recent injury, of brain disease, of poisoning, of heart disease, of paralysis, of kidney disease or of fever. Therefore, we are led at once to the diagnosis of Epilepsy or of Eclampsia. The latter can be excluded by the frequently recurring attacks at long intervals.

If we next take the symptom of clonic convulsion with the aid of chart XIa, we find that there is no fever and the convulsion is a universal one, and not local at the onset. There is coma and there are no symptoms of disease of the brain or cord, or of the kidneys, heart or blood, or of poisoning and thus we confirm the diagnosis of Epilepsy.

Case X. Female, aet. 34.—Nine years ago one morning, her left arm, leg and side of face felt numb and she could not see things on her left side without turning her head. These symptoms steadily increased during the day and she could not use her leg, and especially her arm, well. She could always walk, but at first she could walk only with difficulty. This difficulty in walking gradually passed away. She could use her arm, but could not use it well for more than a year, and it is not quite right even at the present time. The numbness of the left side and the inability to see things on her left still persist. On physical examination there is found anesthesia and analgesia of the left arm and leg and of left side of body and face, (left hemianesthesia and hemianalgesia), also blindness in each eye for all objects to the left of central

vision (left homonymous hemianopia). The left arm and leg are a little awkward and a trifle weak; strength of left hand grasp to that of right is as 80 to 105. Knee-jerks lively, perhaps stronger on left side: neither ankle-clonus nor Babinski. Organic reflexes normal.

Important symptoms: HEMIANESTHESIA, HEMIANALGESIA and HOMONYMOUS HEMIANOPIA.

The principal symptoms of this case are hemianesthesia, hemianalgesia and homonymous hemianopia. These are sensory symptoms and indeed symptoms of a diminution of sensation. We turn to the table of contents and find that "diseases causing a diminution of sensation" are considered in chart XIV, which we next consult. Starting with disorders of sensation in the first column, we have five alternatives offered us in the second column, among which we ought, without doubt, in this case to select diminution of sensation and following this division we have in the next column three alternatives, among which, undoubtedly, we should select anesthesia and analgesia and turn to chart XIVa. In this chart we have the alternatives of the tendon reflexes being either absent or present. In the above case they are present. The dilemma in the next column is quickly decided because the organic reflexes are normal. The history of a motor paralysis lasting a year or more and still slightly persisting directs us to the first alternative in the next column, especially as there are no hysterical symptoms present; while the unilateral nature of the symptoms and next the acute onset (one day) brings us to the diagnosis of Cerebral Hemorrhage or Softening.

To determine which lesion is present, we follow the first cross reference, No. 503-6, which we find in chart Xb. In looking over the abstracts differentiating cerebral hemorrhage, embolism and thrombosis, our case, with its relatively slow onset, its absence of any coma, its absence of any source for an embolism, is probably one of cerebral thrombosis and certainly one of cerebral apoplexy.

The next question is as to the locality of the softening. To ascertain this we turn to the table of contents and find that "localization from symptoms of paralysis" is discussed in chart XXII to which we turn. The reflexes being present in our case, we are brought to the question whether sensory or motor paralysis is dominant. In our case sensory paralysis is dominant and we turn to chart XXIIc. Of the first alternative offered us in this chart we must choose the first: anesthesia and analgesia. In regard to the next column, the distribution of the anesthesia in our case evidently falls into the class: "the (left) arm, leg and face are anesthetic." In our case there is no Jacksonian epilepsy and there is hemianopia, so that the localization of the softening is in the posterior part of the right internal capsule. If we now turn to fig. 17 we can easily see how a lesion in the posterior portion of the internal capsule can easily involve the sensory fibers from one-half the body and also the optic fibers; the continuation of the optic tract. It is also easy to understand that on account of the wide-spread circulatory disturbances in the early stages of the disease, before a collateral circulation had to a degree reestablished itself in the periphery of the lesion, the motor fibers lying directly anterior should be involved and a more or less temporary hemiplegia should occur, as was indeed the case. It seems strange that deafness did not occur in this case as it is certain that the auditory fibers also must have been involved in the lesion, but it is well known that central lesions only produce deafness, even unilateral deafness, when the lesion is bilateral (see page 8).

Thus we have arrived by means of the charts to the diagnosis of this case of "thrombosis of the artery supplying the posterior portion of the internal capsule," but in order to make this diagnosis doubly sure, let us take another one of the prominent symptoms, such as homonymous hemianopia, and follow it through the charts. This symptom is also a diminution of sensation and therefore we turn again to chart XIV. Disregarding this time diminution of sensation we follow "disturbances of vision" and "limitation of field of vision" to chart XIVb. Here we find homonymous hemianopia and in the next column there can be no doubt that we must choose the path which hemianesthesia indicates and by it are led to the diagnosis of hemorrhage, or softening, in the posterior part of the posterior limb of the opposite sided internal capsule, which is the diagnosis to which we had already reached by another road.

CHART X

Motor Paralysis

DIAGNOSTIC ANALYSIS OF SYMPTOMS.

SYMPTOMS ANALYZED	TESTS	
	PERMANENCE OF PARALYSIS	REFLEXES IN PARALYZED MUSCLES
469 MOTOR PAR- ALYSIS OR PARESIS (244) After a careful ex- amination has shown that the paralysis is a true one and is not simulated by any ankylosis or by pain on motion.	470 CONTINUOUS PARALYSIS	Abolition or diminution of both voluntary and reflex acts in the muscles involved. 472 FLACCID PARALYSIS (252) Lesions of peripheral motor neu- rons. There are hypotonia and changes in the electrical reaction of the nerves and muscles involved in very varying degree from simple diminution in excitability, to complete reaction of degenera- tion.
		The differential diag- nosis of those dis- eases in which FLACCID PARAL- YSIS occurs is set forth in CHART X a.
	471 INTER- MITTENT PARALYSIS.	Abolition or diminution of volun- tary, with persistence or even exaggeration of reflex, acts in the muscles involved. 473 SPASTIC PARALYSIS (251) Lesions of central motor neurons. There is hypertonia without altera- tions of electrical reaction of the nerves and muscles.
		The differential diag- nosis of those dis- eases in which SPASTIC PARAL- YSIS occurs is set forth in CHART X b.
		474 A combination of FLACCID PAR- ALYSIS in the upper part of the paralyzed area, and of SPASTIC PARALYSIS in the lower part.
		The differential diag- nosis of those dis- eases in which there is a combination of FLACCID and of SPASTIC PARAL- YSIS, and of those in which INTER- MITTENT PARAL- YSIS occurs is set forth in CHART X c.
		All the muscles of the body and head. The muscles of one or both legs, rarely of arms. Commencing in legs, extending to arms Associated with a cervical rib.

CHART X a
Flaccid Paralysis

Comprising Numbers 475 to 477 on left side of Chart
and 482 to 500 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

472 F L A C C I D P A R A L Y S I S	475 No muscular atrophy, except rarely in chronic cases. Reflexes may be diminished only, not abolished.	Paralysis beginning in the feet and ascending to the head in <i>adults</i> . No true paralysis but great hypotonia in <i>infants</i> .	The paralysis is in the form of a paraplegia, commencing in the feet, and causing death usually in the second year of life. The disease is probably a neuritis (488). The spasm of the dry's paralysis and in addition hematuria. (Compare 488).	Occurs usually congenitally, rarely during the first year of life. No abnormal positions. The child cannot use the slightest movements, no disturbances of organic reflexes. Electrical reaction muscular.
	476 Muscular atrophy, usually great, following the paralysis after the second week of the disease.	The organic reflexes are permanently disordered (1 and 323-4).	Marked sensory symptoms, such as pain, paresthesiae, anesthesia, etc., are present with the motor symptoms. The legs only are paralysed and exhibit trophic disturbances. There is incontinence of urine and the bladder is empty or nearly so.	Very acute onset. Synesthesia. May be blood-stained. Acute, sub-acute or chronic. Other evidence of syphilis. Very chronic and progressive. Very acute, acute or sub-acute of the cord. Fibrillations of the perineum, genitals and
	Paralysis primary.		Sensory symptoms, such as pain, nerve and muscle tenderness, paresthesiae, anesthesia, etc., are present.	Many spinal (very rarely cerebral) nerves are affected.
	The Degenerative Atrophies. (See also Syringomyelia—552, 837-9.)	The organic reflexes are normal or show only transitory disturbances (1 and 323-4).	In very exceptional cases sensory symptoms may be practically absent.	The paralysis is coincident with the distribution of one, rarely of a few spinal nerves.
			No sensory symptoms, except rarely pain and tenderness over the nerve trunks in early stage.	The paralysis is confined to the distribution of one or more cranial nerves.
				Extensor muscles are alone affected.
				A paralysis of acute onset, usually confined to the arms and legs, generally to a portion of one or both; in rare cases involves the cranial nerves.
				A chronic form, may occur exceedingly rarely.
				A paralysis of chronic onset commencing in the peronei muscles and extending symmetrically. Intrinsic muscles of the feet affected.
	477 A combination of muscular atrophy and apparent hypertrophy.	Paralysis secondary.	A chronic disease commencing in childhood or youth and usually showing marked heredity. It exhibits a progressive muscular atrophy, usually combined with some hypertrophy, hence called muscular dystrophy. In time all the muscles become atrophied. The organic reflexes are normal and there are no sensory symptoms whatever and no motor paralysis, except such as would result from the muscular degeneration. Even the apparently hypertrophied muscles are weak. Tendon reflexes are early much diminished and finally absent in the affected muscles. There are no fibrillary contractions. The course of the disease is progressive, but very chronic, lasting many years. From its point of commencement the atrophy extends throughout the body. It produces a marked lordosis. Although the muscular dystrophies are divided into three groups, there are many transitional and mixed forms, and the examination of the excised muscles also shows mixed forms.	The disease is of the extremities. Muscles are atrophied with a mixture of hypertrophy and atrophy. More or less of the disease is present in the extremities. The disease is of the extremities. Muscles are atrophied with a mixture of hypertrophy and atrophy. More or less of the disease is present in the extremities.

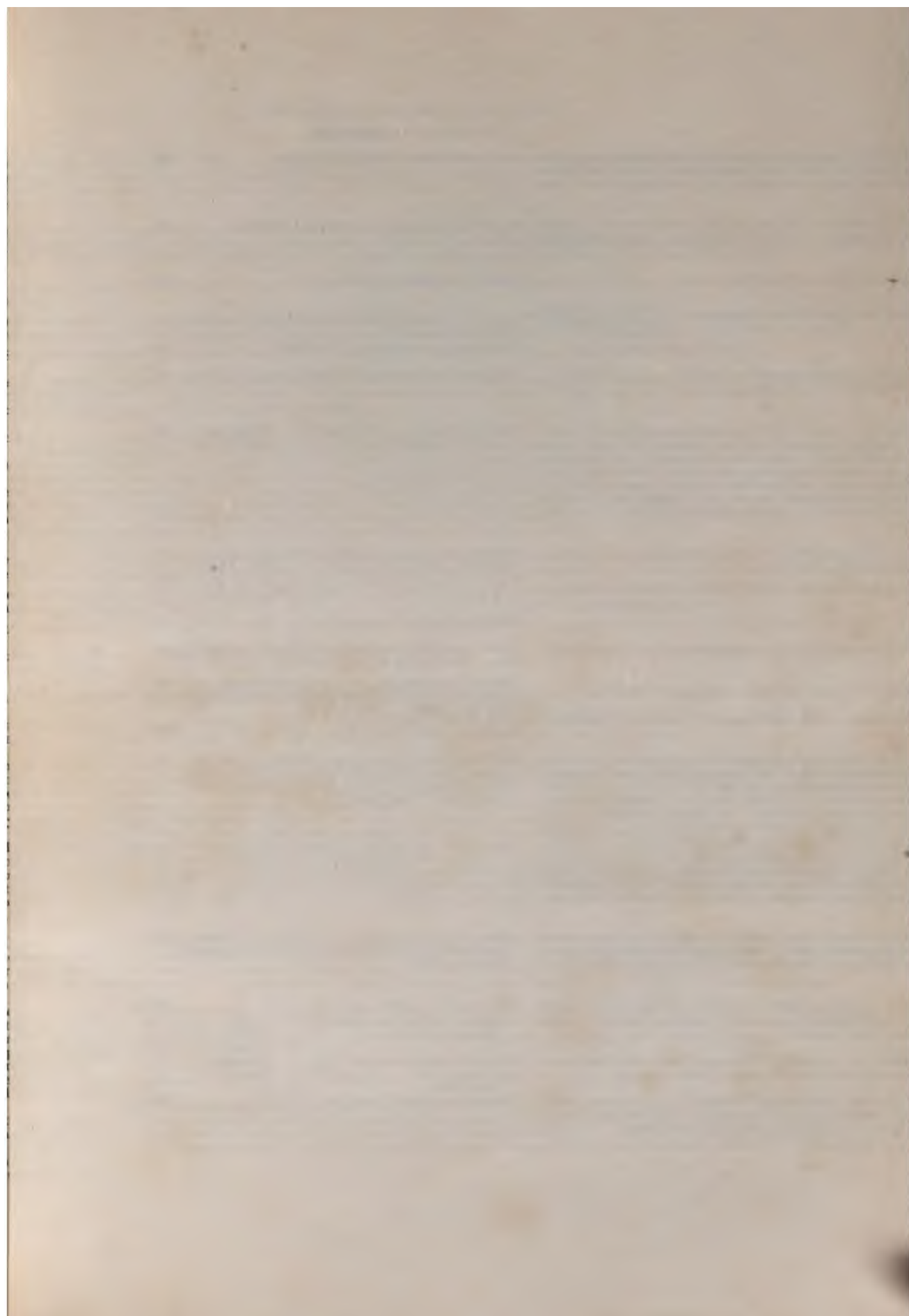




CHART Xb

Spastic Paralysis

Comprising Numbers 478 to 481 on left side of Chart
and 501 to 527 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

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478
Hemiplegia
or Diplegia or
Monoplegia
(254-5, 258)

Congenital or acquired in infancy. There may be fever or apoplectic symptoms at onset.

A motor paralysis of one (infantile hemiplegia) or both sides (Litt) are common and may mask the exaggerated reflexes. In walking and at times idiocy or insanity. Frequently there is a partial of cerebral diplegia, bulbar symptoms (434) are present without expressions, etc., can occur involuntarily, but no voluntary motion.

Almost always in adults and after middle life. Sudden onset, or stroke (ictus), usually with coma (205 and 1037), or with headache or vertigo and mental confusion. Not infrequently the attack commences with a hemiplegia which may or may not be followed by coma.

Symptoms of irritation (convulsions, rigidity, etc.) are more pronounced than are symptoms of paralysis. The d ache

Symptoms of paralysis are more pronounced than those of irritation (convulsions may occur, especially in cortical lesions and in hemorrhage into the ventricles, in which case lumbar puncture may yield a bloody fluid). The paralysis is in part temporary and in part permanent in varying degree. Slow improvement with almost perfect recovery in rare cases. More or less permanent mental impairment, often very slight. Usually patients are more emotional than previously. Exaggerated reflexes and ankle-clonus are present after coma has cleared up. Babinski is present from the start. Puffing, stertorous respiration is common. Cheyne-Stokes' respiration (435) and tracheal rales are very unfavorable symptoms. A sud the balls arte face hem Simila arte mon Simila syph basil hem

479
Hemiplegia, or
Monoplegia
(254, 258)
(See also
Syringomyelia—
552, 837-9.)

Gradual onset without coma, except as a terminal symptom.

Sensory symptoms are always present. Organic reflexes are normal or only slightly disordered.

Brain symptoms. Steadily increasing psychic disorder, and local motor and sensory disturbances over the same area. Head mon mon men usus incre ccm

Spinal symptoms. Paralysis of motion and sensation on opposite sides of body. Charac Sym spin

Choreic symptoms.

The paralysis is only slight and follows or a a paralysis (chorea mollis).

Cranial and spinal nerves are involved.

Intention tremor, nystagmus, scanning speech

Arms and legs are paralysed. Priapism is common, also respiratory difficulty and early death. Radiating pains are common.

There may be a history of injury and a fracture. No history of injury. Little or no pain. Syphilitic myelomalacia (1211). May be history of remote injury. Much pain spinal fluid.

There may be a history of injury and a fracture. No history of injury. Little or no pain. Syphilitic myelomalacia (1211).

May be a history of remote injury. Much pain

Evidence of Pott's disease or tumor compression. In cases of compression due to Pott under increased tension and may contain

History of working under increased atmospheric pressure

Old age, atheromatous arteries, arterial tension

Tumor can be seen or felt on back replacing is involved, or not. Club-foot is common

Signs of irritation predominate over those of paralysis unless the cord is also involved. Usually

Legs mainly involved. Arms involved later and slightly, if at all. These diseases may occur in severe anemia.

Paralysis purely motor, a passive motion, especially with a multiple sclerosis (659). This disease may be simulated

Ataxia. There is a combination of motor and sensory symptoms. In some cases, especially to

480
Paraplegia
(257)
(See also
Syringomyelia—
552, 837-9.)

There is paralysis always of motion and commonly of sensation, usually in the form of paraplegia, more rarely in the form of a spinal hemiplegia (442), which later may become a paraplegia. The reflexes are exaggerated. Ankle-clonus and Babinski are present. Spasms and contractures and bed sores are often present. The organic reflexes are disordered. The motor paralysis is permanent or lasts a very long time. Sensory paralysis may be slight and transitory and may be altogether absent. The anesthesia is often limited above by a narrow zone of hyperesthesia.

481
Paralysis of any extent: local, monoplegia, hemiplegia, or paraplegia

Paralysis limited by some prominent anatomical landmark.

The motor paralysis is usually accompanied by a great variety of the physician (imaginary or delusional paralysis). A paralysed retention of urine is common. Hysterical symptoms (425). In this is sometimes of value in diagnosis.





CHART X_c

Combined and Intermittent Paralysis

Comprising Numbers 471 and 474 on left side of Chart
and 535 to 556 on right margin

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Spinal nerves alone involved.
(Figs. 24-7, 33, 38.)

No sensory symptoms.

Marked sensory symptoms are present, such as pain, paresthesiae, anesthesia, etc., with the motor paralysis.

Dissociation of sensation (365) is present.

Crossed paralysis
(256) and bulbar
symptoms (434).

Chronic—The chronic forms of these diseases, with the spinal form (547) constitute the progressive muscular atrophies and resemble the muscular dystrophies in that the paralysis and atrophy advance together slowly, and it is difficult to say which is primary. They also constitute a group of chronic degenerative atrophies.

Symmetrical paralysis commencing in the small muscles of hands or in shoulder girdle muscles.

Both arms and legs are paralysed. There are trophic disturbances in the arms and not in the legs. Pupils are often unequal. Reflexes are abolished in the arms and increased in the legs. Babinski and ankle-clonus are present. The bladder is usually more or less distended; its detrusor being paralysed. Contractures may be present in the legs.

Dissociation of sensation is the most characteristic symptom and is usually prominent. Pemphigus, ulceration of the skin, and trophic symptoms predominate over motor symptoms in the arms. In the legs the symptoms may be both in arms and legs, and the reaction is exaggerated in central gliosis in the cervical or dorsal regions. The course is fairly rapid course, and may exhibit a unilateral, spastic, muscular.

Paralysis of one or more eye muscles of one eye

Paralysis of facial (both upper and lower branches)

Paralysis of hypoglossus of one side and of other cranial nerves

The onset of paralysis is sudden. If the patient is male, the paralysis is usually unilateral, but may be bilateral. If the patient is female, there is usually a spastic paralysis of the lower extremities. May be due to acute inflammation, hemorrhage, or syphilis. Often due to syphilitic endarteritis.

The paralysis involves the eye muscles.

The paralysis involves the lips, tongue, pharynx and larynx.

The muscles affected show progressive weakness, or, more rarely, in the muscles of the shoulder, the thumb cannot be brought across hand to hand, but not always. There are secondary contractures of muscles is increased. Often associated muscles are involved or not. It is difficult

Very acute onset. Sy may yield a bloody

Acute, sub-acute or chronic
lytic myelomalacia (

Chronic course, intense
common. Cerebro-s

Very chronic onset and

471

INTERMITTENT

Muscles of one or both legs, rarely of arms.

Commencing in legs
extending to arms.

Associated with a cervical rib

The characteristic sign of the disease is the rapid tiring of the muscles when in activity. (401). There is no muscular atrophy and no reaction of degeneration. In the domain usually held retracted on account of the ptosis. The symptoms are slight in the morn

Intermittent attacks of painful muscle cramp, and weakness of leg or legs, caused by wa
Rarely the disease occurs in one or both arms. No sensory disturbances except painful

Recurrent attacks of paralysis of the muscles of the legs usually first and then of arms and nerves are not attacked. There is usually well marked heredity, or the disease occurs but in some groups of family periodic paralysis these negative symptoms are not present.

A cervical rib can be felt and can be seen with the X-ray. In some cases of cervical rib, skin which comes on after the arm has been used a short time, and, if use of the arm is caused by a cervical rib, and then is often relief by elevation of the arm and is ma-

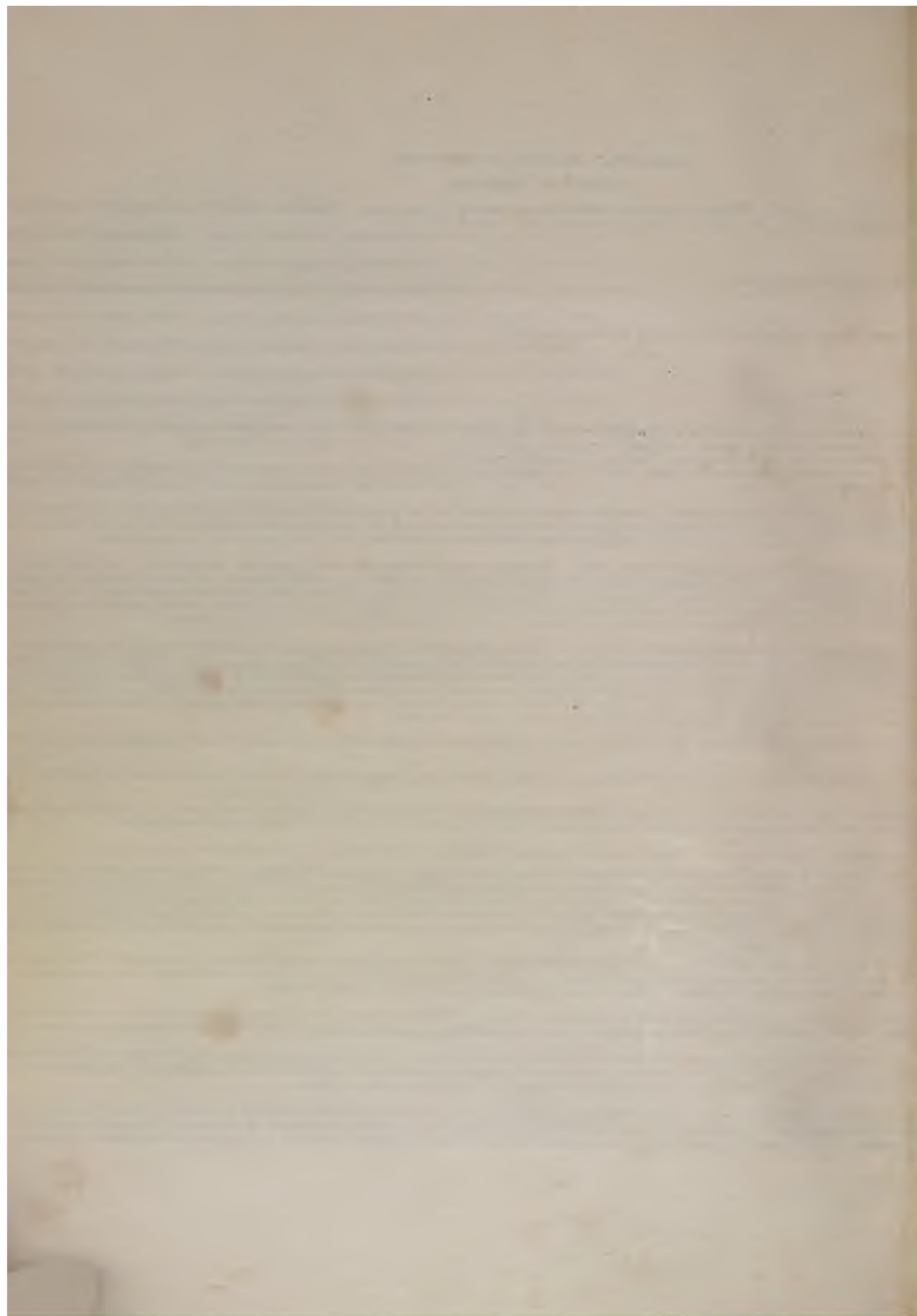


CHART XI

Convulsion or Spasm

DIAGNOSTIC ANALYSIS OF SYMPTOMS

SYMPTOM ANALYZED	TESTS		
	CHARACTER	EXTENT	
570 CONVULSION OR SPASM (242)	571 CLONIC mainly (246)	GENERAL CONVULSION LOCAL CLONIC SPASM	Diseases in which convulsions occur are set forth in Chart XI a.
	572 TONIC mainly (245)	GENERAL TONIC SPASM LOCAL TONIC SPASM	Diseases in which local clonic and all forms of tonic spasm occur are set forth in Chart XI b.
	573 CHOREIFORM (272)	Diseases in which choreiform and athetoid spasm occur are set forth in Chart XI c.	
	574 ATHETOID (271)		

CHART XI a
General Clonic Convulsion

Comprising Numbers 571 on left side of Chart
and 575 to 596 on right margin

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					No other symptom of disease except the convulsion.	{ Repeated attacks. One attack or one series of attacks.
					Symptoms of seri- ous brain disease.	{ Congenital or in infancy. Often fever at onset of first convulsion. In youth or more often in adults.
				Loss of conscious- ness, (coma or semi-coma) (205). Frequently biting of the tongue or other injury. Short duration.	Symptoms of cerebro-spinal disease.	
				The convulsion commences in all the muscles at about the same time (epileptiform convulsion).	Symptoms of dis- ease of other organs than the brain	{ Kidney disease. Cardiac disease. Blood disease.
					Symptoms of poi- soning.	{ Blue line on gums, lead in- toxicity. Alcoholic odor of breath.
571	Apyrexia.			Apparent, but no true, coma (shown by susceptibility to suggestion). No biting of tongue or other injury. Long dura- tion.		Symptoms of hys- teria (425). Such attacks have been called hystero-epi- lepsy.
				The convulsion always commences in one group of muscles and later extends over the whole or part of one side of the body and often over both sides. Jacksonian epilepsy (431).	If the convulsion remains unilateral, con- sciousness may or may not be lost, usually not, but it is always lost when the convulsion becomes bilateral.	
	Hyperpyrexia.	Epileptiform convulsion.			Coma during and after the convulsion.	
				Headache, backache and radiating pains, delirium, vertigo and vomiting, espe- cially on change of posture, hyperalge- sia (spinal and elsewhere), photophobia, etc., are early symptoms. Retraction of head, opisthotonus, etc. (265). Pa- ralysis of cranial nerves (squint, etc.), cutaneous eruptions (herpes), taches cérébrales and Kernig's symptom (319). Tonic spasm and paralysis are more common in basic inflammations, and clonic spasm in cortical inflammations.		{ Lumbar puncture gives a bloody or purulent, fluid pressure containing glo- bulin and many mono- cytes, and if the dis- ease is polymorphonuclear leucocytic. Lumbar puncture gives a normal pressure, fluid containing few cells. Lumbar puncture gives a normal pressure, fluid containing few cells.
	Pyrexia. See also 577.	{ Epileptiform con- vulsion. Coma during and usually after the convulsion.				
				Coma during the convulsion.	{ May occur in children at the onset of any infectious disease, especial- ly diphtheria, scarlet fever, typhoid fever, etc. Is the result of some unusual metabolic changes within the body, as in acute alcoholism, uremia, diabetes mellitus, etc.	

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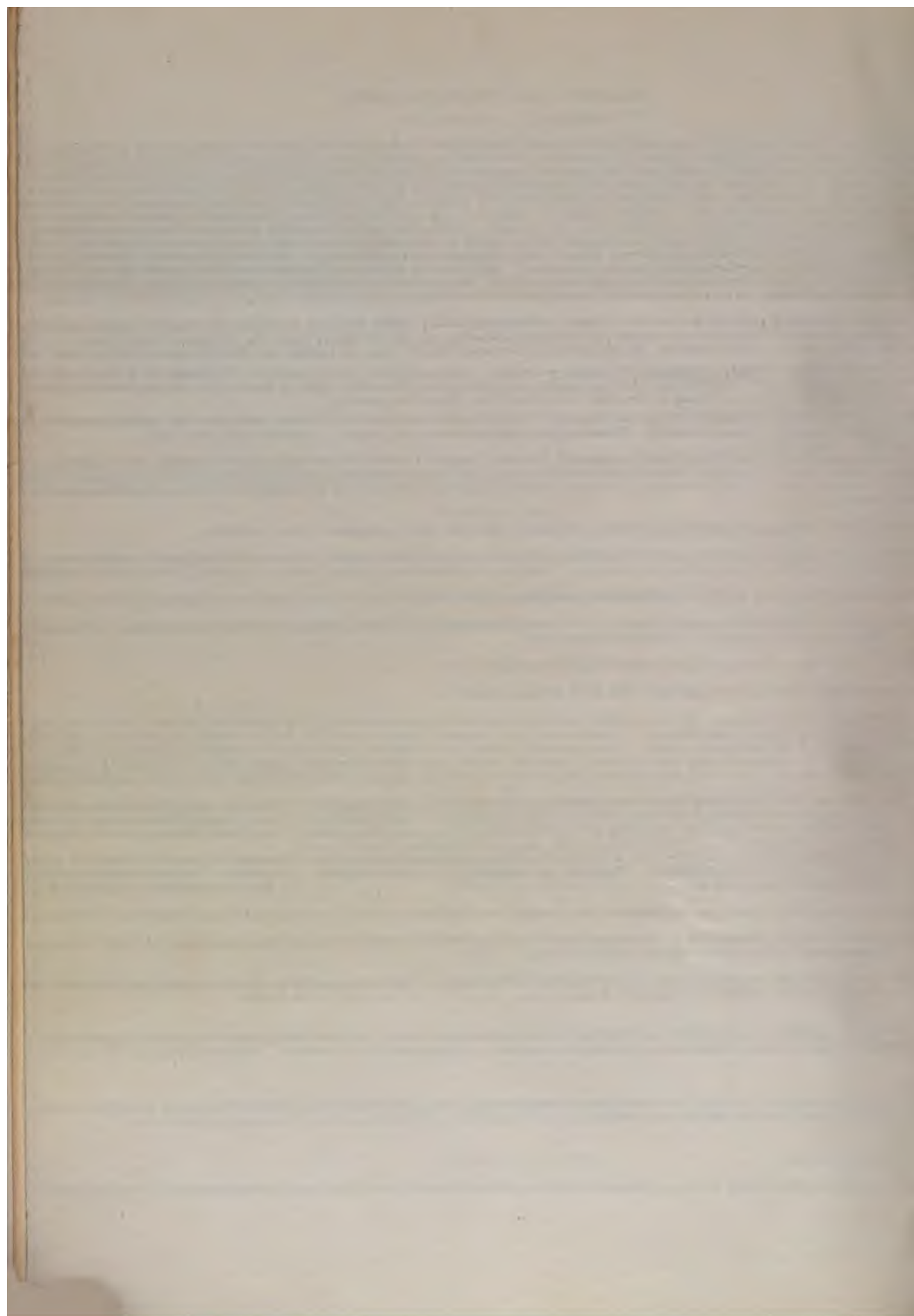


CHART XI b

Clonic or Tonic Spasm

Comprising Numbers 572 on left side of Chart
and 600 to 621 on right margin

(Note)—Many of the spasms, especially the tonic spasms, are associated with pain, and are then called "cramps."

DIAGNOSTIC SYMPTOMS AND TESTS

571	CLONIC SPASM	LOCAL	Pyrexia in very acute cases.	Shock-like spasms similar to that produced by an electric shock.	Begins in one arm side and then to
		CLONIC	Apyrexia	A single or many times repeated spasm, rarely contracture, of one muscle or of a group of muscles, occurring in paroxysms which rather tend to subside on voluntary movements. Myoclonus. (270.)	Occurs in face and more rarely in neck and arms.
		SPASM		Begins in arms and may extend to legs but almost never to face. Often the tendons play as in subsultus tendinum	
		SPASM		Begins in side of face or in one arm or leg and may extend over one, or even both sides of body.	
	GENERAL TONIC SPASM	Pyrexia	Spasm commencing in jaws.	There is the history of an infected wound of jaws, occurring in paroxysms; also body being held in position of opisthotonus becomes very high. The disease varies	
			Spasm commencing in pharynx and oesophagus.	There is history of a bite by an animal, especially on sight of water. Spasmodic cough, opisthotonus and general spasm light and accommodation. The stage diagnosis must be made in such cases	
			Spasm commences in back of neck and in back.	There may be no symptom. Lur	
		Apyrexia. If unconsciousness is present. See also epilepsy media (575).	Cerebellar ataxia is present (281).	A tonic spasm of sudden onset, the face not being affected.	
			Rigidity rather than spasm, not strong enough to prevent passive or voluntary movements (266).	Extremities and trunk attack may last Rigidity of all muscles	
			Spasm only at commencement of any action.	Spasm passes away as the action is continued. The muscles of the face usually escape. Fibers show marked hypertrophy. Closes exposure to cold with consequent. The so-called acquired form, "myoton	
			Spasm mainly confined to hands and feet, paroxysmal.	Bilateral painful tonic spasm of muscles. Increased mechanical (Trousseau) associated with rickets or digestive disorder. It occurs in infectious diseases, in poi	
		LOCAL	General painful clonic, followed by tonic spasm.	Spasm very general. nine poisoning.	
			General permanent contracture.	Paralysis is coincident.	
			Spasm only occurs when performing some accustomed act.	Occurs usually in small muscles and in the gradual onset, steadily grows worse, and rather than spasm. Atrophy of the neck the voice of singers, public speakers, e	
	Apyrexia.		Rather brief spasm of one or more muscles.	A spasm lasting in cough, oesophagus	
		More permanent spasm.	A contracture of a few or many muscles. efforts are made to overcome it. No n bling (674) or may consist in jumpin		
		A permanent or, at least, a long continued, contracture. The muscles are anatomically shortened, in later stages.	A hemiplegic contracture. A paraplegic contracture. A local contracture.		
			Tendon reflexes are and lasts for years Tendon reflexes are follows an attack Absence of reflexes traction of tendons		

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CHART XI_c
Choreiform and Athetoid Spasms

Comprising Numbers 573 and 574 on left side of Chart
and 622 to 631 on right margin

DIAGNOSTIC

CHOREIFORM

ABSTRA

DIAGNOSTIC SYMPTOMS AND TESTS

573 CHOREIFORM SPASM (272)	G E N E R A L	TRUE CHOREIFORM MOVEMENTS	{ Widespread spas- modic contractions of muscles of body generally.	Irregular, quick, involuntary, spontaneous contractions, group of muscles throughout the body or limited to one. Patient is restless and fidgety. Speech is explosive. tarily. The movements have somewhat the character resemble more closely extreme restlessness: grimaces, ti of hands and feet, etc. Some muscular weakness (cho and almost always marked hypotonia or atonia (39, 23 are interfered with and made incoordinate by the occur untary contractions (ataxia). The part cannot be held may be slight, or so strong as to prevent walking or e during sleep, but to some extent prevent sleep. They a excitement. Reflexes are normal but the knee-jerk may back only slowly. Paresthesiae and anesthesia rarely
		PSEUDO- CHOREA	{ Limited to one group of muscles.	{ Involuntary, often unconscious, or unnoticed, execution hemming, winking, etc. Each person has his own ind
			{ Sudden, lightning- like contractions of groups of muscles.	{ The spasms are painful and instantaneous; the platys especially in Northern Italy. In the later stages epile related to chorea minor but is more allied to myoclon
			{ A coarse tremor rather than chorei- form movements.	{ Usually limited to one extremity. Rhythmical trembling of an extrem electric shocks. Other symptoms (273), are purely hysterical and
574 ATHETOID SPASM (271, 504)	L O C A L	A slow contraction of one set after another of small muscles of the hand; rarely of the foot (mo frequently also involved and are usually held contracted in extreme flexion. Squirming, twisti and hyperextension predominating. The athetoid spasm is increased by voluntary movements of The face and neck muscles may not infrequently be affected in bilateral athetosis. The extren always weak but never paralyzed. The spasm may be unilateral or bilateral. Usually in hands, m ments, though slow, are powerful and at times cause sub-luxation of joints. Decided muscular rig		

OF SYMPTOMS

ETHETOID SPASM

SYMPTOMS

and then of another body (hemichorea). These are made involuntary movements, but of tongue, twisting (510) is present; involuntary movements of these involuntary movements. They cease on observation and the foot sink

Common in children, rare in adults. Slight mental disturbances often present. Usually acute, rarely chronic, frequently recurrent. Often associated with rheumatism and endocarditis, rarely with pregnancy (chorea gravidarum).

Occurs only in adults. There is much and progressive mental impairment. Movements coarser and more violent. Heredity. Chronic.

Occurs only in old persons with atheromatous arteries and brain symptoms. Speedy death usually.

Occurs in hemiplegia, (after apoplexy, etc.) and is confined to the incompletely paralyzed extremities, especially the hand and arm. It is most frequent in the hemiplegias of childhood.

act at short intervals. Little "tricks" which characterize many persons such as: coughing, or habit and rarely varies from it. Usually occurs in neurasthenics.

ido-mastoid and hypoglossus muscles are especially affected. It is a rare disease, occurring in convulsions and paralyzes with atrophy occur. Often fatal. This disease is probably not at all

a intensity. At times so coarse and irregular as to resemble chorea, at other times more like present (425). The extensive convulsive movements sometimes called chorea magna or major are in their nature.

Ankles and wrists fingers, extension of the other hand. Activities involved are feet. These movements present.

The athetoid spasm is present from birth. It is very rarely unilateral, more frequently bilateral. There is much mental impairment, even idiocy.

Present from birth or infancy. Some mental impairment. Unilateral or bilateral. Associated with a mild hemiplegia or diplegia. Rare.

Occurs in adult life after an attack of apoplexy. Usually unilateral. Rare.

DIAGNOSIS

Sydenham's, or Infectious, Chorea. 622
Chorea Minor (272).

Huntington's, or Hereditary, Chorea 623
(103).

Senile, or Degenerative, Chorea. 624

Post-hemiplegic Chorea. 625

Habit Chorea or Habit Spasm (274). 626

Electric Chorea. Dubini's Disease 627
(600).

Rhythmical, or Hysterical, Chorea 628
(273).

Congenital Athetosis. 629

Athetosis after cerebral palsy of childhood. 630

Athetosis after apoplexy. 631



CHART XII

Perversion of Motion and Local Palsies and Spasms

DIAGNOSTIC ANALYSIS OF SYMPTOMS

SYMPTOM ANALYSED	CHARACTER	
635 PERVERSIONS OF MOTION (243)	638 ATAXIA (248)	The diseases in which ataxia occurs are set forth in Chart XII a.
	639 TREMOR (250)	
	640 NYSTAGMUS (291)	The diseases in which tremor, nystagmus, or fibrillation occurs are set forth in Chart XII b.
	641 FIBRILLARY CONTRACTION OR FIBRILLATION (292)	

LOCAL PALSIES AND LOCAL SPASMS

636 LOCAL PALSIES	See Chart XII c.
637 LOCAL SPASMS	See Chart XII d.

CHART XII a

Ataxia

Comprising Numbers 638 and 642 to 644 on left side of Chart
and 648 to 664 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

63: A T A X I A (248)	642 Ataxia mainly upon standing or walking. Staggering gait. Static ataxia. Cerebellar ataxia. (281).	No loss of muscle sense. No motor paralysis, except in late stage of 651-2.	Occurs at any age, usually in adults. Usually sensory symptoms.	Sight and hearing normal.
			Occurs in youth. No sensory symptoms.	Sight or hearing abnormal.
	643 Inability to stand or walk. More or less complete.	Bilateral.	Many sensory symptoms.	Occurs in family group though less pronounced is common and
		Unilateral. (Hemiataxia.)	Often analgesia and thermic anesthesia.	Evidently functional.
		Unilateral. (Hemiataxia.)	Loss of muscle sense and sensory symptoms usually prominent. Knee-jerk usually increased.	Evidently organic.
				When of acute course usually follows an apoplectic arterial disease is present.
	644 Ataxia of all movement. Dynamic ataxia. Motor ataxia (280).	Bilateral	Knee-jerks normal.	No ankle-clonus.
			Exaggerated knee-jerks, ankle-clonus and Babinski.	Great variety of local are intention tremor. Rarely the essential point is the
				A combination of symptoms usually lost before Knee-jerks may be
			Knee-jerks and ankle-clonus absent. No Babinski. Often loss of muscle sense and retardation of conduction of pain.	Rarely any permanent ball) are not uncommon held well apart and symptom (448), Argys sensations and paresthesia in patches and in cerebro-spinal fluid. Positive Wasserman mainly affected. In and the diagnosis is
				Slight motor paralysis normal. Cranial nerves never so chronic as
		Irregular distribution.	Knee-jerks usually exaggerated, but no Babinski or ankle-clonus. Evidently functional (pseudo-ataxia).	

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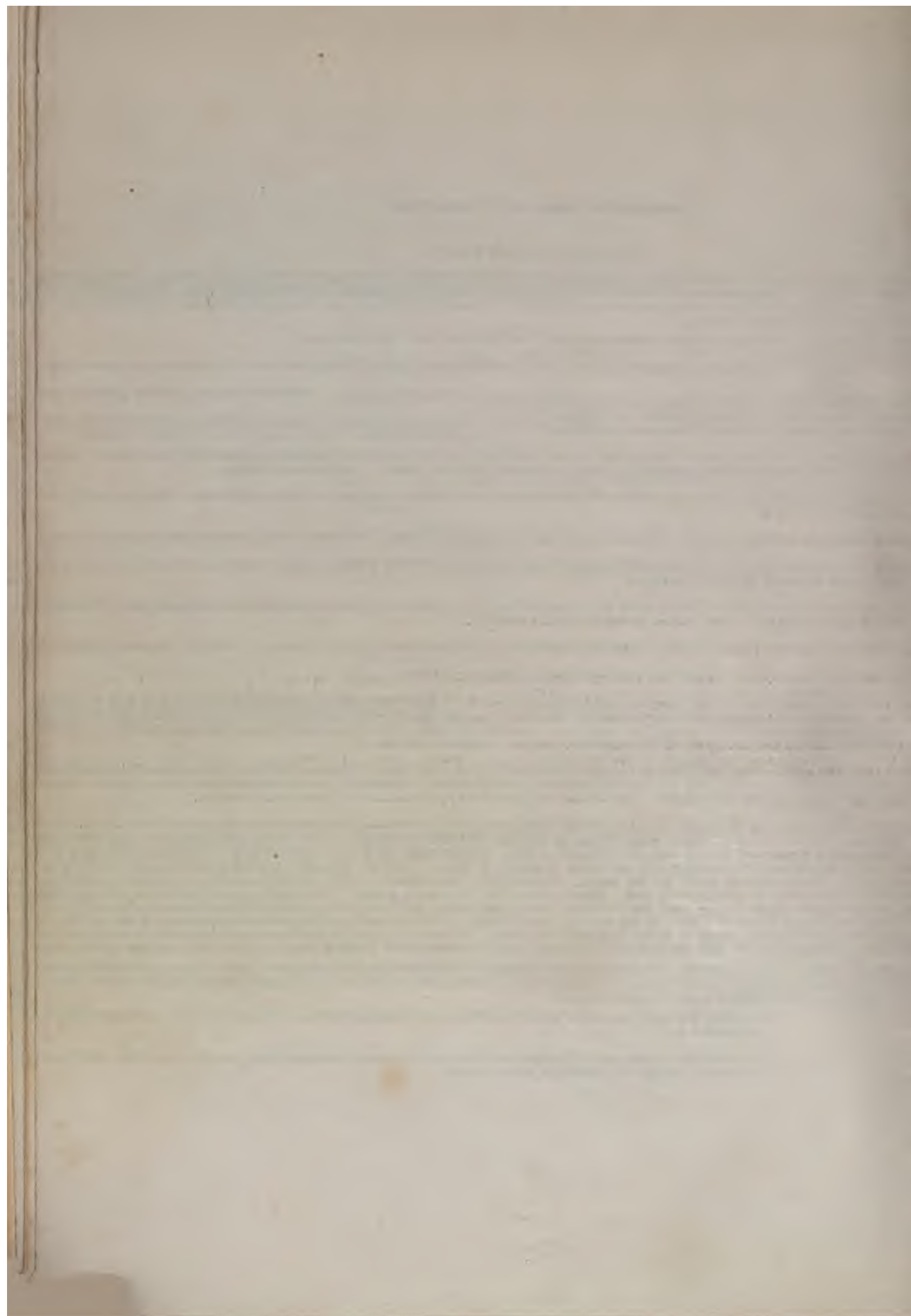


CHART XII b
Tremor, Nystagmus, Fibrillation

Comprising Numbers 639 to 647 on left side of Chart
and 668 to 697 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

639 T R E M O R (250)	645 Intention Tremor (290).	{ Coarse, irregular tremor; 4 to 8 per second. Fine tremor.	{ Tremor is usually associated with scanning speech, usually a great variety of motor and sensory symptoms with their loss, over a very variable amount. Vertigo is a very common symptom. Occurs in family groups and shows well marked heredity. Staggering gait. Ataxia. Nystagmus is common and speech often defective. Tremor is associated with general weakness or conv	{ Occurs Occurs	
	646 Passive Tremor. Increased on voluntary motion and excitement (289).	{ Fine, rapid tremor; 8 to 12 per second. Slow tremor; 3 to 6 per second.	{ Exophthalmus, goitre, tachycardia, vascular throbb when patient looks downward (Graefe's symptom). History of addiction to alcohol or drugs. Mental s greatly at different times. Presence of hysterical symptoms (425). Tremor is Tremor is marked in face, lips and tongue. Progre cytolysis and globulin in cerebro-spinal fluid. Was Slow tremor of hand and foot of same side, associat	{ Occurs Occurs	
	647 Passive Tremor. Diminished on voluntary motion (289).	{ Slow, fine tremor; 3 to 6 per second. Slow, coarse tremor.	{ Tremor, which is associated with muscular rigidity an involves the other side. The tremor is most marka characteristic attitude (head and body bent forward, ilar tendency to run backwards (retropulsion). T The disease often commences with a stiffness and no sensory symptoms except the sensation of rigi Tremor begins bilaterally. Head is early affected. Rotatory or nodding tremor of head occurring sudd involved. The tremor ceases when the child's ey A series of jerky tremors limited to the back, or in Not associated with other nervous symptoms. Her	{ Occurs Occurs Occurs Occurs Occurs	
	Either Intention or Passive Tremor.				
	640 N Y S T A G M U S (291)	Always a symptom of organic disease. Very rarely, an hysterical clonic spasm may simulate true nystagmus (pseudo-nystagmus). This is often vertical and is more rapid and more violent than nystagmus and is associated with other hysterical symptoms (425).	{ No weakness of any rectus muscle. No impairment of sight.	{ Defective vision from whatever Due to lack of pigment in iris Workers in mines. Due to v Vertigo is a prominent symptom. Coarse, jerky tremor is a prominent symptom. Ataxia is also present. Cerebral symptoms present. Rickety baby in winter. Congenital.	{ Paroxysmal violent Vertigo, Occurs i Occurs i Occurs i Most m ing of Lateral
			{ Weakness of one or more of the recti muscles.	{ Marked sensory symptoms. No sensory symptoms.	{ Analgesia Muscula Muscula Muscula Occurs
641 FIBRILLARY CONTRACTION OR FIBRILLATION (292).		{ Evidence of organic disease. Degeneration of peripheral motor neurons. Evidence of functional not organic diseases.	{ Marked muscular atrophy with muscular weakness. No muscular atrophy or weakness.	{ Marked sensory symptoms. No sensory symptoms.	{ Analgesia Muscula Muscula Muscula Occurs



CHART XII c

Local Palsies

Comprising Numbers 700 to 721 on right margin

(Note)—The anesthesia accompanying these palsies can be seen from the areas of cutaneous distribution of these nerves depicted in the plates at the end of the book (Figs. 33-8). In mild lesions of the nerves anesthesia is either absent or much less marked and less extensive than the motor paralysis.

DIAGNOSTIC ANALYSIS OF SYMPTOMS

LOCAL PALSIES

ABSTRACT OF SYMPTOMS

INABILITY TO
MOVE, MORE
OR LESS,
MUSCLES OF
THE

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There are ptosis and strabismus divergens and the pupil is dilated and immobile both to light and accommodation (this condition of the pupil may occur as an isolated paralysis,—333). The eyeball can be moved in no direction except outward (abducens), and outward and downward with rotation of eyeball (superior oblique). For symptoms characteristic of the isolated paralysis of each ocular muscle see Chart XIV c, 816. When the superior oblique muscle is paralysed the levator palpebrae superioris is paralysed with it and ptosis results.

The ocular muscles, except the levator palpebrae superioris, have a bilateral cortical representation. Hence ocular paralyses, except ptosis, almost never occur in lesions above the oculomotor nucleus, except in bilateral lesions. The cortical representation of the ocular muscles seems to be very diffuse or multiple. Conjugate deviation may result from supra-nuclear lesions.

For the symptoms of paralysis of the trochlearis (patheticus) nerve and of the abducens nerve, each of which produces a strabismus convergens, see 816.

DIAGNOSIS
Paralysis 70
of Motor
Oculi.
(Figs. 14, 18)

Paralysis 70
of Trochlearis and
of Abducens

Paralysis 70
of motor
branch of
Trigeminus.

J
A
W

The muscles of mastication of one side, rarely of both sides, are paralysed and in severe cases atrophied. The temporal and masseter muscles cannot be felt firmly contracting when efforts are made to chew. The jaw cannot be closed tightly or opened strongly or moved laterally towards the healthy side (external pterygoids), or the chin pushed forwards (internal pterygoids). Mastication of food is difficult or impossible; dysmasesis (286). The jaw reflex (322) is abolished. In some cases one side of the soft palate (tensor veli palatini) is paralysed and in some the hearing of low tones is unpleasant (tensor tympani).

In trigeminus lesions there is unilateral abolition of the conjunctival, corneal, sneezing and palatal reflexes; and the secretion of tears is at times affected. There is no irritation, or tears, from inhaling ammonia or acetic acid. There is also loss of sense of taste, and dilatation of the pupil, narrowing of the eyelid slit, even enophthalmus, are present. Heat and redness of skin in recent cases and coldness and cyanosis of skin in old cases. The salivary secretion and taste are affected when either the proximal or the distal end, but not the middle, of the nerve is affected.

F
A
C
E

The muscles of expression of one side (rarely of both sides) of the face are paralysed. The forehead cannot be wrinkled and the eye appears larger than normal and cannot be closed (lagophthalmus, hare's eye). When attempts are made to close the eyelids the eyeball turns upward, the cornea disappearing behind the upper lid (Bell's phenomenon). The angle of the mouth is lower than normal and cannot be raised. The naso-labial fold is obliterated. The lips cannot be firmly closed; so that whistling is impossible and speech is impaired. Mastication is difficult because the weakened buccinator muscle allows food to collect between the jaws and the cheek. The platysma is also paralysed; so that the angle of the mouth cannot be drawn downwards. Tears may flow from the eye and irritate the cheek and saliva from the angle of the mouth. The conjunctiva may become inflamed and the cornea ulcerated, because the eyelid cannot wink and keep the conjunctiva clean. In some cases the facial paralysis may be preceded and accompanied by pain. In severe cases the paralysed muscles exhibit the electrical reaction of degen-

Facial 70
Paralysis.
Bell's
palsy.
Prosopoplegia.
Facial
Monoplegia.
Facial
Diplegia,
(751, 928,
1317).

LOCAL PALSIES (Continued)

ABSTRACT OF SYMPTOMS

DIAGNOSIS

INABILITY TO
MOVE, MORE
OR LESS,
MUSCLES OF
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eration. Hearing and taste are frequently impaired and disordered. When taste is affected the salivary secretion is also affected. In the early stages of the disease the face is drawn over toward the healthy side by the unantagonized healthy muscles. In the later stages the face may be drawn back again permanently towards the paralyzed side by the contracting newly formed connective tissue in the degenerated muscles. Also in the early stage of recovery the face may be drawn towards the paralyzed side by over-innervation of the muscles formerly paralyzed, and may exhibit temporary contractures and spasms, possibly "associated movements." These spastic symptoms may be due to irregular regeneration of the nerve. The upper fibres of the facial nerve have a bilateral cortical representation as do the laryngeal nerves. Hence lesions of the cerebral hemispheres paralyse mainly the lower branch of the facial; the eye on the paralysed side can be closed but is easily forced open. For the localization of the different forms of facial paralysis, see 1317.

Paralysis of the pneumogastric nerve is discussed under 760. In addition to the laryngeal paralysis there is often present disorder of the respiratory act and of the heart beat (tachycardia).

Pneumo- 704
gastric
Paralysis
(760).

When the tip of the shoulder sinks downwards and forwards and the arm cannot be easily raised, there may be a paresis of the trapezoid muscle. When this muscle is paralysed on both sides, the head tends to fall forward. When the head is drawn towards one shoulder and the chin turned upwards and towards the other, the sterno-cleido-mastoid muscle is paralysed on that side toward which the chin turns. This posture is called caput obstipum spasticum, when the muscle is atrophied and secondarily contracted and the deformity can no longer be corrected by passive motion. Caput obstipum spasticum occurs also and is more pronounced in torticollis from spasm of the muscle (730). When the sterno-cleido-mastoid muscle is paralysed on both sides, the head tends to fall backwards.

Paralysis 705
of the
Spinal
Accessory.

N
E
C
K

The tongue when protruded turns towards the paralysed side. When both sides are paralysed the tongue cannot be protruded at all, and in such cases, speech, mastication and deglutition are difficult and imperfect. In lesions of the *nucleus* of the hypoglossus nerve there is also a mild paresis of the orbicularis oris muscle. Intracranial lesions involving the hypoglossus and other nerve roots at the base of the brain may cause Avelli's syndrome: pharyngo-laryngeal or glosso-pharyngo-laryngeal paralysis (546); or may cause Schmidt's syndrome: the above and also sterno-cleido-mastoid and trapezius paralysis.

Hypo- 706
glossus
Paralysis.
(546, 752).

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The diaphragm is paralysed on one or both sides, causing dyspnoea on exertion and sinking in of the epigastrium on inspiration, especially on deep inspiration. The lower part of the lung is drawn upwards and atelectasis and pneumonia may occur. Besides the usual causes of compression and neuritis, this paralysis may also occur in pleurisy, peritonitis, trichinosis and in bulbar and spinal lesions. The paralysed diaphragm shows Litten's phenomenon.

Phrenic 707
Paralysis.

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A
G
M

The supra- and infra-spinatus muscles are paralysed; so that rotation of the arm outward and raising it in abduction are impaired. Muscles involved are atrophic and ulnar side of hand is turned forwards.

Supra- 708
Scapular
Paralysis.

A
R
M

The serratus anticus major is paralysed; so that when the scapula is raised, its lower angle approaches the vertebrae and the inner margin of the scapula does not lie close to the thorax and, on movements of the arm upwards and forwards, stands from the thorax like a wing. The arm cannot be raised beyond a horizontal line.

Long 709
Thoracic
Paralysis.
Serratus
Paralysis.

LOCAL PALSIES (Continued)

ABSTRACT OF SYMPTOMS		DIAGNOSIS
INABILITY TO MOVE, MORE OR LESS, MUSCLES OF THE A R M (C o n t)	Motion of the arm inward and forward is impaired. Hand cannot be placed on opposite shoulder.	Anterior and Posterior Thoracic Paralysis. 710
	Rotation of the arm inward and motion of the arm backward are impaired.	Sub-Scapular Paralysis. 711
	The deltoid and teres minor are paralysed: so that the arm cannot be raised.	Axillary Paralysis. 712
	The combined paralyses of the brachial plexus: Erb's and Klumpke's paralysis, are discussed under 454, 455 and 490.	
H A N D	The biceps, brachialis anticus and coraco-brachialis muscles are more or less completely paralysed; so that flexion of the arm at elbow is more or less impaired, especially in supination (very rare).	Musculo-Cutaneous Paralysis. 713
	The pronators and flexors of the hands and fingers, the muscles of the ball of the thumb and the first and second lumbrical muscles are paralysed. The hand can neither be flexed nor pronated. The thumb cannot be brought across hand to touch the little finger, but remains close to the index finger (ape's hand). The first (proximal) phalanges of fingers can be flexed, but not the second and third phalanges.	Median Paralysis. 714
	The interossei, the third and fourth lumbricals, and the muscles of the little finger are paralysed. The proximal phalanges cannot be flexed, the other phalanges cannot be extended and the little finger cannot be moved. The fingers cannot be spread. When muscle atrophy and contracture occur "claw hand" results.	Ulnar Paralysis. 715
	The extensors and supinators of the hand and fingers, and the abductor pollicis longus, are paralysed. The thumb is adducted and can neither be abducted nor extended. Wrist-drop and slight pronation. Wrist and fingers cannot be extended completely. The wrist-drop differs from that of lead palsy (494) in that the supinator longus is paralysed. Therefore, if the forearm is held midway between supination and pronation and the elbow strongly flexed against a resistance offered, the belly of the supinator longus will not stand out firmly contracted as it will in lead paralysis and in health.	Musculo-Spiral and Radial Paralysis. 716
L E G	The extensor femoris is paralysed; so that flexion of the thigh on the body and extension of leg on thigh are impossible or difficult. Standing and walking are difficult, and ascension, jumping and running impossible.	Crural Paralysis (997). 717
	The adductor muscles of thigh are paralysed; so that adduction of leg, pressing of thighs together and crossing of legs are impossible.	Obturator Paralysis. 718
	The glutei muscles are paralysed; so that walking, ascending stairs, straightening up of body, abduction and rotation of thigh are impaired. Generally much muscular atrophy.	Gluteal Paralysis. 719
	Foot and toes are paralysed; the leg cannot be flexed on thigh and rotation of the thigh is impaired. In cases of isolated tibialis paralysis there is absence of plantar flexion of foot, and of plantar, flexion, spreading and adduction of toes (Pes calcaneus et valgus). In cases of isolated peroneal paralysis there is absence of dorsal flexion and abduction of foot and its adduction impaired—absence of dorsal flexion of toes. There are foot-drop, high stepping gait and Pes equino-varus.	Sciatic Paralysis. (996) 720
	For paralysis from lesions of the cauda-equina, see 487, 1007 and 1308.	Cauda Equina Paralysis (Fig. 29). 721

CHART XII d

Local Spasms

Comprising Numbers 725 to 733 on right margin

DIAGNOSTIC ANALYSIS OF SYMPTOMS

LOCAL SPASMS

SPASM OF MUSCLES OF		ABSTRACT OF SYMPTOMS	DIAGNOSIS
J A W	{	The jaws are held tightly shut and the masseter and temporal muscles can be felt to be contracted (lock jaw), usually bilaterally. The spasm may be "tonic," as in tetanus (606), tetany (614), irritation of teeth (wisdom teeth) and certain unilateral lesions of the pons and medulla; or "clonic," as in chills and in rare cases of paralysis agitans and hysteria. When the pterygoid muscles alone are in spasm the mouth is held open and cannot be closed.	Trigeminal Spasm or Cramp. Trismus. 725
F A C E	{	Spasms of one or more muscles of expression of the face, unilateral or bilateral, are relatively common, as in convulsive tic (601) and tic douloureux (602). These spasms are often a mixture of tonic and clonic contractions, the clonic predominating. They may affect all the muscles or only one, as in tonic spasm of the orbicularis palpebrarum (blepharospasm) (601, 617), or in clonic spasm of this muscle (spasmus nictitans: nictation). The platysma myoides often takes part in these spasms and very rarely the muscles of the soft palate and the internal and external ear muscles. Very rarely spasm of some of the facial muscles about the mouth constitute an occupation neurosis or cramp, as in the "Auctioneer's cramp" and "Cornet player's cramp." These facial cramps may be symptomatic directly of lesions of the cortical facial center, of the facial nerve in its course, and reflexly of the trigeminal nerve or its terminal filaments in the eye, nose, mouth or ear. There is also to be remembered the passive contracture of the degenerated muscles and the active contracture due to over-innervation of the convalescing muscles in facial paralysis. Causeless and uncontrollable laughter must also be classed among the facial spasms. This condition, similar to the allied state of causeless and uncontrollable crying, occurs especially in hysteria and in lesions of the optic thalamus.	Facial Spasm or Cramp (267, 601). 726
P H A R Y N X	{	Spasm of the pharynx of a tonic nature preventing swallowing and of a clonic nature repeating the act of swallowing with great frequency occur. The former occurs in hydrophobia (607) and somewhat also in tetanus (606); while the latter, associated with coma, frequently occurs in mild epileptic attacks. The spasm also occurs from irritation of the pharynx in hysteria and very rarely, as one of the crises in locomotor ataxia (433). Spasm of the oesophagus is not uncommon in hysterical persons and makes the swallowing of food very difficult.	Glossopharyngeal Spasm or Cramp. 727
L A R Y N X	{	Spasm of the muscles of the larynx (spasmus glottidis, false croup, laryngismus stridulus), causing noisy and difficult breathing, is a not uncommon and occasionally a dangerous condition. It occurs almost exclusively in children and is often associated with rickets and with digestive disorders. Occurs also in general diseases such as hydrophobia, hysteria, epilepsy, chorea, tabetic crises, etc. Sneezing (sternutatio spastica, ptarmus) and coughing, reflex acts implicating both the pneumogastric and the intercostal nerves, are often due to pathological conditions and irritation of the nervous system. Bradycardia, Cheyne-Stokes' respiration and cerebral vomiting are symptoms of irritation of the pneumogastric nucleus, but are not characteristic and are of little diagnostic value.	Pneumogastric Spasm or Cramp. 728
T O N G U E	{	Spasm of the tongue is very rare, especially so the tonic form. During the attack speaking and swallowing is impossible. Very rarely a tonic spasm of the tongue occurs when the patient attempts to speak (stuttering and aphthongia). Spasm of the tongue is sometimes associated with facial spasm and with spasm of the submaxillary muscles. These spasms may be due directly to lesions of the cortical tongue center, of the hypoglossus nerve in its course, or reflexly, especially from lesions of teeth, mouth and nose.	Hypoglossus Spasm or Cramp. 729

LOCAL SPASMS (Continued)

SPASM OF MUSCLES OF

ABSTRACT OF SYMPTOMS

DIAGNOSIS

N
E
C
K

{ Spasm of the neck muscles, especially the sterno-cleido-mastoid: caput obstipum (spastic wry neck), is sometimes congenital and is sometimes acquired in later life. In these cases the head is drawn toward the shoulder of the affected side and the chin is turned toward the other side and slightly elevated and the sterno-cleido-mastoid muscle can be felt to be firmly contracted. When the trapezius is the seat of the spasm the occiput is drawn backwards and turned toward the shoulder of the affected side and the edge of the muscle can be felt to be firmly contracted. Spasm of the muscles is sometimes tonic, sometimes clonic and often both. The cause of these spasms is often neurotic and often rheumatic. Rarely it is some disease of the eye or of the ear (torticollis ab oculo laeso, ab aure laesa) or of the cervical vertebrae. Usually many muscles are involved, although one or two more prominently than the others.

Spinal 730
Accessory
Spasm or
Cramp
(601).

D
I
A
P
H
R
A
G
M

{ Tonic spasm of the diaphragm, either unilateral or bilateral, occurs very rarely and produces dangerous dyspnoea. It sometimes occurs as one symptom of a general disease: tetanus, hydrophobia, hysteria, etc. Clonic contractions are common and cause hic-cough (singultus), always a distressing and at times a dangerous symptom, which occurs occasionally in brain and spinal cord lesions and frequently in irritation of the pneumogastric nerve, especially from the gastric mucous membrane. A similar but slower contraction of the diaphragm associated with facial spasm (opening of mouth) causes the act of yawning (oscedo, chasmus) which is sometimes frequently repeated as an aura of apoplexy or epilepsy and occurs also in hysteria, digestive disorders, drowsi-ness, etc.

Phrenic 731
Spasm or
Cramp.

A
B
D
O
M
E
N

{ Tonic and clonic contractions of some or all of the abdominal mus-cles occur with extreme rarity, and are usually, if not always, hysterical.

Inter- 732
costal
Spasm.
Abdominal
Spasm.

A
R
M

A
N
D

L
E
G

{ Tonic and clonic spasms of the muscles of the arm and shoulder or of the leg, with the exception of the secondary contractures due to lesions of the pyramidal tract and of the peripheral nerves, are very rare. They usually are due either to deficiency of water in the system, and often occur in disease in which much water is lost, as cholera, diarrhoea, etc., or to hysteria, or to rheumatic factors, or are reflex. The deformity resulting in each case can be predicted from the function of the muscle involved.

Brachial, 733
or Lumbar,
or Sciatic
Plexus,
Spasm or
Cramp.

CHART XIII

Disorders of Speech and Gait

DIAGNOSTIC ANALYSIS OF SYMPTOMS

SYMPTOMS ANALYSED	CHARACTER OF DISORDER	
735 DISORDERS OF SPEECH, READING AND WRITING.	737 ANARTHRIA (283) Inability or unwillingness to speak. No disease of vocal organs or peripheral nerves. This condition may result from a complete aphonia (260) or complete aphasia (221) or complete dysarthria (284).	The diseases in which Anarthria and Dysarthria occur are set forth in Chart XIII a.
	738 DYSARTHRIA (284) Ability to express thought by speech but articulation is defective.	
	739 APHASIA (221) Articulation normal but expression of normal thought is defective.	
736 DISORDERS OF GAIT.	740 ATAXIC	The varieties of Aphasia and the conditions under which they occur are set forth in Chart XIII b.
	741 PARALYTIC AND FLACCID	
	742 PARALYTIC AND SPASTIC	
		The diseases in which Disorders of Gait occur are set forth in Chart XIII c.

CHART XIII a
Anarthria and Dysarthria

Comprising Numbers 737 and 738 on left side of Chart
and 743 to 768 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

737 A N A R T H R I A (283)	Result of disease in infancy, or congenital.	Auditory memories necessary for understanding spoken words were never acquired, or were early lost through disease; hence innervation memories necessary for speech were never learned.		{ May make noises but cannot speak Can be trained to speak through sight.	
	Result of disease in adult life.	Innervation memories necessary for speech have been acquired but are not available. No hysterical symptoms.		{ Complete absence of speech, and is impossible. Patient makes no sound or to communicate by gesture. Will neither whisper nor speak. Can whisper faintly but distinctly. May mutter but cannot articulate	
		Hysterical symptoms and etiological factors present, although not always prominent.			
		Apoplectic symptoms (504).			
	Congenital.	Vocal organs defective.		{ Words imperfectly formed, also a nasal voice. At times words are not understood. Words imperfectly formed and usually a very limited vocabulary. Substitution of one letter for another. An examination of the speech shows the vowels correctly but has difficulty in pronouncing the consonants.	
		Vocal organs normal.			
	Defective Education.	Vocal organs normal.		{ Patient cannot whistle or close lips tightly. Tongue is not protruded straight but deviates to one side. Soft palate is not raised (bilateral) or not raised symmetrically. Anesthesia of larynx. Paralysis of crico-thyroid muscle (on lower level) and of thyreo-ary-epiglottis muscles. Immobility of one or both vocal cords from paralysis. In normal position of cords (between extreme adduction and abduction) in cases of unilateral paralysis, the healthy cord is paralysed.	
	738 D Y S A R T H R I A (284, 1388)	Paralytic.	No symptoms of any central disease.		{ Immobility of one or both vocal cords from paralysis. Cord or cords lie near the median line (extreme adduction) and become smaller on inspiration. Immobility of one or both vocal cords from paralysis. Cord or cords lie near the median line (extreme adduction) and become smaller on inspiration.
		The labials, the linguals or the vowel sounds or all of them cannot be properly pronounced. A careful examination reveals a paralysis or a paresis within the domain of the facial, the hypoglossal or the pneumogastric nerve.		{ Immobility of one or both vocal cords from paralysis. Cord or cords lie near the median line (extreme adduction) and become smaller on inspiration. Immobility of one or both vocal cords from paralysis. Cord or cords lie near the median line (extreme adduction) and become smaller on inspiration.	
		May be symptoms of central disease.		{ Unilateral or bilateral paralysis of the soft palate, all the laryngeal muscles and anesthesia of larynx.	
		Tremor and Ataxia.	Slow and clumsy speech.	Cerebellar gait.	{ Speech sounds as if a foreigner. Argyll-Robertson's phenomenon attacks may occur. Childlike appearance. Alcoholic history, appearance of disease.
			Tremulous and slovenly speech, words are badly formed, letters and syllables are left out both in speaking and writing.	Evident mental deterioration.	
		Rigidity.	Scanning speech.	Intention Tremor.	{ Great variety of widespread rigidity of muscles and slight tremor.
			Monotonous speech.	Passive Tremor.	
Spasm.		Certain letters (consonants) are spoken with difficulty and are repeated many times imperfectly.		{ Utterance is arrested by a spasm of one or more of the muscles concerned in speech, such as the larynx, pharynx, or tongue. Singing is usually not at all affected.	



THE HISTORY OF THE
CITY OF BOSTON

From its first settlement in 1630 to the present time. By SAMUEL JOHNSON, Esq. of the Middle Temple, Barrister at Law. In two Volumes. The first Volume contains the History from 1630 to 1780. The second Volume contains the History from 1780 to the present time. With a Plan of the City, and a Map of the Harbor. Boston: Printed and Sold by S. KNEELAND, at the Sign of the Anchor, in the South Church Yard, 1789.



Printed and Sold by S. KNEELAND, at the Sign of the Anchor, in the South Church Yard, 1789.

CHART XIII b
Amnesia and Aphasia

Comprising Numbers 739 on left side of Chart
and 769 to 777 on right margin

TEST

739

AMNESIA AND APHASIA
(221 to 227)

None of these conditions constitutes a disease, but is rather one symptom of a more complex disease. Each is a form of dementia in the broad sense of the term and consists in a loss of general or special memories. See also Anarthria and Dysarthria (738-8).

Patient is capable of normal speech but exhibits a decided loss of memory.

The loss of memory may not be accompanied by any, or of the names of persons is rather common and of no distant past, are referred by the memory to the cerebral concussion and compression (1042-3), especially little time immediately previous to the injury and fright.

Examination of the patient shows a loss of memory, especially in old people and in the insane, and is usually associated

Patient is incapable of normal speech for want of innervation memories of a few or many spoken words.

Can express ideas by gestures, but cannot name objects when it is spoken to him and can often then pronounce one or two words, or even to none (anarthria). Uses and often even when it is not. Cannot construct sentences or sing songs. When his arm is not paralysed patient can usually read but not aloud. The condition is usually

Patient is incapable of normal speech for want of auditory memories of spoken words.

Patient fails to understand more or less of what is said to what he repeats. Cannot execute verbal commands, but conscious of this mistake even when his attention is called

Patient is incapable of normal speech for want of visual memories of written or printed words.

Patient cannot name objects seen, or read written or printed. Cannot execute written commands, but readily executes Can write from dictation imperfectly, but not at all from

Patient is incapable of normal speech from loss of innervation memories and of auditory memories of spoken words.

Patient cannot at a loss called to

Patient is incapable of normal speech from loss of proper associations and of appreciation of the memories concerned in speech.

Patient cannot exhibits

AGRAPHIA.

Patient's speech is normal, but his writing is abnormal.

Patient is incapable of writing for want of the necessary condition uncomplicated by motor aphasia.

Patient omits words in writing, uses the wrong words, mis

Broca, in 1861, published a case of motor aphasia with a lesion at the base of the left inferior frontal convolution and thereby laid the foundation for the study of aphasia. Wernicke (whose studies have contributed greatly to the comprehension of aphasia) divided motor and sensory aphasia into three

- 1st. Cortical Motor Aphasia, in which the patient is unable to speak, write or read aloud correctly, or to speak or write correctly.
- 2nd. Sub-cortical Aphasia, in which the patient can neither speak spontaneously nor from dictation nor read aloud correctly.
- 3rd. Transcortical Motor Aphasia, in which the patient can neither speak nor write correctly, but can speak and write from dictation.
- 1st. Cortical Sensory Aphasia, in which the patient can speak (with paraphasia) and copy, but can neither write, nor speak, nor read.
- 2nd. Sub-cortical Sensory Aphasia, in which the patient can speak quite perfectly, write, copy, read aloud and understand written words.
- 3rd. Transcortical Sensory Aphasia, in which the patient can speak (with paraphasia) and write (with paraphasia), can copy, read and understand written words.

Wernicke also recognizes a Conduction Aphasia, in which the patient can speak, write and read and understand correctly, but exhibits

Marie considers all forms of aphasia as resulting from a greater or less degree of a general intellectual impairment rather than to local lesions of the brain (aphasia with difficulty of articulation (anarthria or dysarthria). Whether he is altogether right in this or not, certainly our conceptions of aphasia, neither of which is probably altogether false.

ANALYSIS OF SYMPTOMS

APHASIA AND AGRAPHIA

LIST OF SYMPTOMS

DIAGNOSIS

only by very little, intellectual impairment in other respects. To a certain degree the loss of memory has no prognostic or prognostic value. "Retroactive amnesia" is where events, which occurred in the more immediate past, as in Korsakoff's psychosis (1100). "Retrograde amnesia" occurs in some cases of those associated with fright. In it, memory is lost of those events which occurred during some	Amnesia.	7
usually for recent events, impaired judgment and a general failure of mental powers. Very common and with mental depression.	Dementia (1077).	7
well, or at all. Can use verbs better than nouns and proper names. Recognizes the desired word in it. In speaking, the patient is frequently at a loss for a word. His vocabulary is limited often to a wrong word (paraphasia—775) but is often conscious of his mistake if his attention is called to it correctly (agrammatismus), but can often repeat sequences of numbers, days, months, etc., and can usually write from copy, but makes many mistakes in spontaneous writing (paragraphia—777). Usually associated with right-sided hemiplegia in right-handed persons and vice-versa.	Motor Aphasia or Aphemia (221, 1390).	7
him. Cannot repeat what is said to him, or if in rare cases he can do this, he does not understand and readily executes written ones. In speaking, the patient frequently uses a wrong word and is not called to it. Can write spontaneously and from copy but not from dictation. He can read well.	Sensory Aphasia. Auditory Aphasia. Word Deafness (222, 1345).	7
and letters or words, but may at times recognize and name objects which he touches and feels. Verbal ones. In speaking, patients rarely use a wrong word and are conscious of their mistakes. Can copy and make many mistakes in spontaneous writing. Cannot read what they have written.	Visual or Optic Aphasia. Alexia. Word Blindness (228, 1391).	7
neither name objects nor understand words spoken to him. In speaking, patient is frequently wrong for a word or uses a wrong one and is then unconscious of his mistake, even when attention is called to it. He may or may not be able to read and writing is impossible or very defective.	Mixed Aphasia (224).	7
puts words in speaking, uses the wrong word, puts words in a wrong place in the sentence and incoherent, even jargon speech.	Paraphasia (225).	7
conservation memories. His arm and hand are not paralyzed for other movements. A very rare	Agraphia (227, 1389).	7
puts up words in the sentences so that writing becomes incoherent.	Paragraphia (226).	7

foundation, not only of the modern ideas about the faculty of speech, but also of cerebral localization.

divisions each:

from dictation, or to read with full understanding, but can copy correctly and understands what is said to him.

that can read, write and understand what is said to him.

dictation, can copy, can read aloud, and can understand speech and writing.

can copy from dictation, nor read aloud perfectly, nor understand speech or writing.

copy, but cannot speak or write from dictation, nor understand speech.

write, and speak from dictation, and read aloud, but all without understanding, and cannot understand either speech or writing.

paraphasia and paragraphia.

cerebral lesions, especially not to those of the left inferior frontal convolution. He considers motor aphasia to be a combination of sensory aphasia previously to Marie's article had been growing too schematic. The truth probably lies somewhere between Marie's and Wernick



CHART XIII c

Disorders of Gait; Ataxic, Paralytic and Flaccid, Paralytic and Spastic Gaits

Comprising Numbers 736 to 742 on left side of Chart
and 780 to 800 on right margin

(Note)—In addition to the diseases mentioned in this chart, pain, whether in the joints (rheumatism, gout, arthritis, morbus coxae, etc.), or in the muscles (rheumatism, myositis, etc.), or in the bones (caries, etc.), or in the nerves (sciatica, etc.) will cause a limping gait. The gait of a patient suffering from weakened arches in the feet is very characteristic in advanced cases and this common disease should always be thought of in any disturbance of gait and in any painful affection of the legs or lower back.



CHART XIV

Disorders of Sensation

ANALYSIS OF SYMPTOMS

SENSATION AND OF THE SPECIAL SENSES

DISORDERS IN SENSATION

- | | | | |
|-----|--------------------------------------|---|------------------|
| 810 | Anesthesia and Analgesia. | } | See Chart XIV a. |
| 811 | Dissociation of Sensation. | | |
| 812 | Loss of Muscle Sense. | | |
| 813 | Hyperesthesia. | | |
| 814 | Perversion. | } | See Chart XIV b. |
| 815 | Limitation of field of vision. | | |
| 816 | Double vision. | } | See Chart XIV c. |
| 817 | Conjugate Deviation of Eyeballs. | | |
| 818 | Pupillary Abnormalities. | } | See Chart XIV d. |
| 819 | Ophthalmoscopic Examination. | | |
| 820 | Deafness (anakusia). | } | See Chart XIV e. |
| 821 | Hyperakusia (oxyakoia) or Parakusia. | | |

mell.

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CHART XIV

Disorders of Sensation

DIAGNOSTIC ANALYSIS OF SYMPTOMS

DISORDERS OF GENERAL SENSATION AND OF THE SPECIAL SENSES

SYMPTOM ANALYSED	ALTERATIONS IN SENSATION		
804 Disorders of Sensation. (See also Perversion of Sensation Chart XV).	805 Diminution of Sensation.	810 Anesthesia and Analgesia.	See Chart XIV a.
		811 Dissociation of Sensation.	
		812 Loss of Muscle Sense.	
	806 Exaggeration of Sensation.	813 Hyperesthesia.	
		814 Perversion.	See Chart XIV b.
		815 Limitation of field of vision.	
	807 Disturbances of Vision.	816 Double vision.	See Chart XIV c.
		817 Conjugate Deviation of Eyeballs.	
		818 Pupillary Abnormalities.	See Chart XIV d.
		819 Ophthalmoscopic Examination.	
	808 Disturbances of Hearing.	820 Deafness (anacusia).	See Chart XIV e.
	809 Disturbances of Taste and Smell.	821 Hyperakusia (oxyakoia) or Parakusia.	

CHART XIV a
Disorders of Sensation

**Comprising Numbers 805 to 812 on left side of Chart
and 822 to 841 on right margin**

DIAGNOSTIC SYMPTOMS AND TESTS

805 DIMINUTION OF CUTANEOUS SENSIBILITY (345).	810 ANESTHESIA usually com- bined with some ANALGESIA and THERMIC ANESTHE- SIA, especially in severe cases of the disease (348-50). (Figs. 26, 33).	Tendon reflexes diminished or absent (lesion of peripheral sensory neurons—472).	Organic reflexes normal (300). (Figs. 24-6).	T
			Organic reflexes disordered (300). (Figs. 24-7.)	A N
		Tendon reflexes normal or ex- aggerated in arms or legs or both (lesion of central sensory neurons—473).	Organic reflexes disordered; it may be only slightly (300). (Figs. 19-30.)	A A N
			Organic reflexes usually normal, very rarely disor- dered (300).	A m A hy to
		Tendon reflexes absent in arms; exaggerated in legs. Lesion both of peripheral and of central sensory neurons.	Organic reflexes slightly disordered (300).	A
806 EXAGGERATION OF CUTANEOUS SENSIBILITY.	811 ANALGESIA and THERMIC ANESTHESIA with little or no TACTILE ANESTHESIA (DISSOCIATION OF SEN- SATION) (365).	Tendon reflexes usually exag- gerated in legs (473). Organic reflexes little or not at all disordered (300).	Arms affected. Legs affected. Motor paralysis and hyp opposite side of the bo	P P P
	812 AKINESTHESIA.	Loss of muscle sense is usually associated with ataxia and parietal cortex.		
	812a NUMBNESS.	Unilateral numbness of hand and foot, steadily progressive Bilateral numbness is of no diagnostic importance. It m		
806 EXAGGERATION OF CUTANEOUS SENSIBILITY.		Hyperesthesia and hyperalgesia are of little or no diagnos		
		myelitis.		

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CHART XIV b

Disturbances of Vision

Comprising Numbers 807, 814, 815 on left side of Chart
and 842 to 866 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

807
DISTURBANCES
OF VISION814
PERVERSION.

A yellow color of all objects seen irrespective of their true color; xanthopsia.

A red color (erythropsia) of all objects seen irrespective of their true color (erythropsia).

A green color of all objects seen irrespective of their true color (green vision).

Muscae volitantes, twisted threads and irregular spots moving about in field of vision.

Flashes of light and dark spots surrounded by a bright zone (glittering scotomata).

Achromatopsia (364) and hemichromatopsia occur in slight lesions of the geniculate ganglion.

An inversion (red having a larger field than the blue—14) and an interlacing, of the color fields (Dyschromatopsia).

{ Hysterical symptoms (425) are present. Choked disc and other symptoms of increased intra-cranial pressure is present.

Blindness (358, 1318). No lesion within orbit.

{ Bilateral.
Unilateral or Bilateral.

No lesion in eye. Pupillary reflex normal.

{ No lesion in eye. Optic neuritis present.

{ No lesion in eye. No optic neuritis shown that the blindness is not hysterical.

Homonymous Tetartanopia or Quadrant Hemianopia.

No hemiopic pupillary reflex. No or other paralysis. May or may not be choked disc. Very rarely occurs in lesion of optic fasciculus of opposite side.

815
ABSENCE OR
LIMITATION
OF FIELD OF
VISION
(358 to 364).

Homonymous hemianopia (14, 362, 1321). May very rarely be bilateral, due to double lesion.

{ No hemianesthesia. No hemiopia (26). No hemianopia.

{ Hemianesthesia.

{ May or may not be a hemiopic pupillary reflex. Paralysis of abducens nerve or both.

Bitemporal hemianopia (362, 1319).

Nasal hemianopia (362, 1320).

{ Slow onset, progressive course, terminates in complete blindness. Choked disc and pupillary reflex.

Horizontal hemianopia.

Occurs in lesions of the retina, or optic chiasm.

Homonymous scotomata.

These may occur as the result of lesions in the neighborhood of the calcarine fissure.

Concentric limitation of field of vision, even to complete blindness.

{ Increased tension of eyeball. Exophthalmos.

{ No increased tension of eyeball. Or normal.

{ Hysterical symptoms (425) are present.

{ Symptoms of tabes are present, especially no ataxia. History of Syphilis.

OF SYMPTOMS

VISION

SYMPTOMS

on).

Seen especially when eyes are turned towards a bright light.

denly appearing and disappearing in the field of vision.

s, of the optic fasciculus and especially of the calcarine cortex.

sease are present. The color field becomes normal after the in-
(Cushing.)

Uremic amaurosis may be in this class (edema).

ent. Pupillary reflexes absent.

ary reflexes normal. Hysterical symptoms. By tests it may be

esthesia { Upper homonymous quadrant of each field of vision.
choked { Lower homonymous quadrant of each field of vision.
tract

reflex { Sudden onset and of short duration. Often more
alysis. { marked in, or limited to, one eye. No other symp-
{ toms except nervousness. Circulatory disturbances.
{ Choked disc. Slow onset. Progressive course of the
{ disease.
{ No choked disc. Rapid onset. Permanent, not pro-
{ gressive, or rarely shows a regressive course.

No hemiopic pupillary reflex. No choked disc. Re-
gressive course.

miopic { Choked disc. Slow onset. Progressive course.
uli or { No choked disc. Rapid onset. Symptoms of menin-
{ gitis may be present.

usually { Bilateral.
miopic { Unilateral

rve or chiasm, involving their upper or lower portion.

ns in the geniculate bodies, in the optic fasciculus or in the

and final atrophy of optic nerve. Pupils dilated and unequal.
disc.

oscopic examination the optic papilla shows atrophy.

gyll-Robertson's phenomenon and absence of reflex. Little or
and lymphocytosis in cerebro-spinal fluid.

DIAGNOSIS

Jaundice, or Santonin, Amyl Nitrite, Cannabis 842
Indica or Picric Acid Poisoning.

Neurasthenia, Hysteria, great emotional excitement 843
and after cataract operations; also after the eye
has been exposed for a long time to an electric or
other bright light (snow-blindness).

Diseases of optic nerve and retina and after cataract 844
operations.

Neurasthenia, circulatory disturbances in brain and 845
digestive disturbances.

Migraine, and Aura of Epilepsy, and circulatory 846
disturbances in brain.

Achromatopsia 847

Hysteria (1074). 848

Cerebral Tumor (833). 849

Lesion or edema of both occipital lobes. 850

Lesion of optic nerve or chiasm. 851

Hysterical Amblyopia 851a

Lesion of lower lip of contralateral calcarine fissure. 852

Lesion of upper lip of contralateral calcarine fissure. 853

Aura of migraine. 854

Tumor involving median surface of contralateral 855
occipital lobe or fasciculus of Gratiolet (1364).

Hemorrhage or softening in or near contralateral cal- 856
carine fissure or optic fasciculus of Gratiolet (1364).

Hemorrhage or softening in the posterior part of 857
posterior limb of contralateral internal capsule.

Tumor involving contralateral optic tract or genic- 858
ulate bodies (893, 1321).

Neuritis or lesion of contralateral optic tract (893, 859
1321).

Tumor compressing central part of optic chiasm 860
(892, 1319). (Enlarged pituitary.)

Tumor compressing homolateral outer part of optic 861
chiasm (892, 1320).

Horizontal Hemianopia 862

Homonymous Scotomata 863

Glaucoma (943). 864

Optic atrophy. (898.) 865

Hysteria (1074). 866

Tabes (661). 867

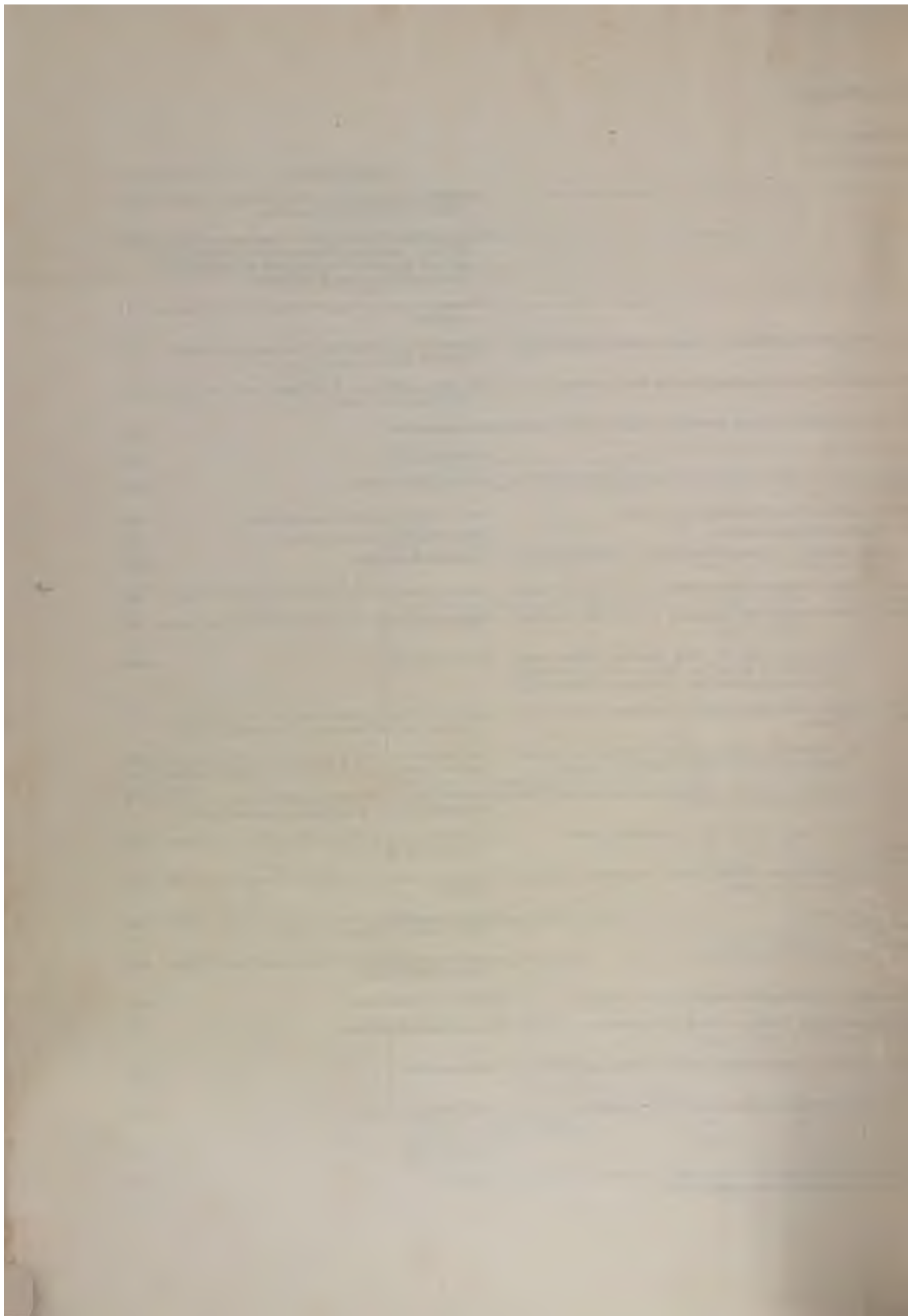


CHART XIV c

Disturbances of Vision

DIAGNOSTIC ANALYSIS OF SYMPTOMS

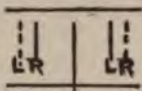
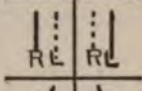
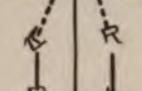
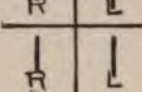
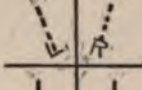
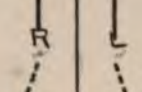
CHARACTER OF THE DIPLOPIA	SECONDARY DEVIATION OF SOUND EYE (29)	DISPLACEMENT OF VISUAL AXIS (28)	LIMITATION OF MOTION	POSITION OF FALSE IMAGE (SEE 28)	GRAPHIC REPRESENTATION OF THE DIPLOPIA. BROKEN LINE IS THE FALSE IMAGE	DIAGNOSIS
BINOCULAR	Inward.	Inward. Strabismus convergens.	Outward.	On the same side as the affected eye.		Ex- 870 ternal Rectus.
	Outward.	Outward. Strabismus divergens.	Inward.	On the opposite side to the affected eye.		In- 871 ternal Rectus.
	Upward.	Downward. Strabismus deorsum vergens, slightly divergens.	Upward and somewhat inward.	Above and on opposite side to the affected eye, image tilted top inward.		Su- 872 perior Rectus.
	Downward.	Upward. Strabismus sursum vergens, slightly divergens.	Downward and somewhat inward.	Below and on opposite side to the affected eye, image tilted top outward.		In- 873 ferior Rectus.
	Downward and inward.	None or slightly upward and inward. Strabismus sursum vergens, slightly convergens.	Rotation downward and somewhat outward.	Below and on same side as the affected eye, image tilted top inward.		Su- 874 perior Oblique.
	Upward and inward.	None or slightly downward and inward. Strabismus deorsum vergens, slightly convergens.	Rotation upward and somewhat outward.	Above and on same side as the affected eye, image tilted top outward.		In- 875 ferior Oblique.
MONOCULAR	Absent	May be variable.	The limitation of motion and the position of the false image are the reverse of those in paralysis. There may be present some irritation, especially in the nose or teeth, which would cause a reflex spasm. The spasm is usually more transient than a paralysis. The muscles usually affected are the internal rectus and the inferior oblique.		Fig. 14	Spasm of the 876 ocular muscles.
	The whole eyeball can be seen to be displaced.		Displacement of eyeball.			877
	No changes visible in eye.		Hysterical symptoms (425) are present.			Hysterical 878 diplopia.
	Changes visible in eye.		Two openings can be seen in pupil.			Double pupil- 879 lary opening.
OF VISION	By oblique illumination the lens can be seen to be opaque in patches.		Cataract.			880
	Examination shows dislocation of lens.		Dislocation of lens.			881
	Examination shows astigmatism and an irregular contour of the cornea.		Irregularities of cornea.			882
	Associated with other symptoms of lesions in the pons. Eyes turned away from the side of the lesion. Deviation is usually not present when the eyeballs are at rest. A vertical deviation of the eyeballs occurs very rarely. It is associated with a lesion of the corpora quadrigemina. (1271).		Lesion near the anterior portion of the pons (cephalad) to the abducens nucleus, involving posterior longitudinal bundle.			883
817 Conjugate deviation of eyeballs.	Eyes turned to the side of the lesion.		Paralytic lesion in almost any part of brain, especially, in posterior part of frontal lobe.			884
	Eyes turned away from the side of the lesion.		Irritative lesion in cerebral cortex.			885

CHART XIV d

Abnormalities of Pupil and Optic Papilla

Comprising Numbers 818, 819, 890, 891, 897 and 898 on left side of Chart
and 890 to 914 on right margin

PUPILLARY ABNORM

DIAGNOSTIC SYMPTOMS AND TESTS

ABSTR

818
ABNORMALITY
OF PUPIL

Disordered pupillary reflex to light and accommodation (330-1).
Mydriasis, myosis or unequal pupils (339-41).

These phenomena.
Their significance

890
The hemiopic
pupillary reflex.
(26).

Bitemporal hemianopia (362, 1319).
Homonymous hemianopia (362, 1321).

Choked disc. Syphilis.
Often hemiplegia (history of syphilis).

891
The Argyll-Robertson's
phenomenon (447).

History of syphilis. Lymphocytosis
in cerebro-spinal fluid.
Positive Wassermann.

Ataxia. Absence of
Mental impairment.
Rarely occurs. No

Albumen and casts

Sugar in urine and

Lead in urine.

Examination of t
shows a condition
anemia.

Urine and blood no

Retinitis. No marked symptoms of cerebral disease.

No retinitis. Marked cerebral symptoms.

Bilateral.

Unilateral.

897
Optic neuritis.
Choked disc.

Well marked history.
Increased size of h
Retraction of head.

General convulsion
lepsy is common
paralysis. Re
creased.

819
ABNORMALITY
OF PAPILLA.

Result of
Ophthalmoscopic
Examination.

898
Optic atrophy.

Bilateral.

Unilateral.

Secondary. It may be the terminal stage of a
Traces of the active inflammation

Primary. No signs
of a former inflam-
mation.

Old age. Usually atheromatous a
Loss of knee-jerk. Myosis. Light
Unequal pupils. Impairment of sp
Childishness.
Characteristic tremor or other sym

Local inflammation or lesion can usually be made out or

ANALYSIS OF SYMPTOMS

PROGRESSIVE OPTIC NEURITIS AND ATROPHY

SYMPTOMS

Many conditions to be of much diagnostic importance. Discussed in Chart Vb.

Progressive, terminating in blindness. Often associated with acromegaly.

of cranial nerves. Optic neuritis or symptoms of meningitis. At times a field defect or a quadrant hemianopia in partial lesions of the geniculate bodies.

Lightning pains. Girdle sensation and tabetic cuirass.

Speech. Apraxia. Restlessness. Childishness. Uncontrollable.

Seizures present. May be no mental impairment. Normal speech. No apraxia.

Headaches, especially in morning. Usually edema of some part of body. Dyspnoea on exertion and loss of strength.

Progressive emaciation and loss of strength. Great thirst and polyuria. Large appetite. Dry skin.

Blue line on gums. History of lead colic. Wrist-drop. History of exposure to lead poison.

Dyspnoea on exertion and progressive weakness. Pallor of skin and mucous membranes.

History of syphilis. Argyll-Robertson's pupillary reflex. Lymphocytosis in cerebro-spinal fluid. Positive Wassermann.

in which the nerve has been injured. Usually complicated with facial paralysis.

Scars and sutures open in the young.

Local lymphocytosis. Fever.

epi-
local
in- { May or may not be fever. At times a latent period. Primary suppuration of bones of skull or elsewhere. Optic neuritis present in about 53% of cases.

{ No fever. Usually steady progression. Optic neuritis present in about 80% of all cases; almost invariably present in tumors in the posterior fossa. Tumors in pituitary gland, corpus callosum and in the central convolutions, especially extra-cerebral tumors, often show no optic neuritis.

DIAGNOSIS

Tumor compressing the optic chiasm (851, 860-1, 1319-20). 892

Lesion of contralateral optic tract or geniculate bodies (858-9, 1321). 893

Tabes (661, 827). (Figs. 24-7.) 894

Paresis (1104). 895

Syphilis (1205). 896

Bright's Disease. 899

Diabetes Mellitus (1175). 900

Lead Poisoning (494, 576, 584, 788, 988, 1050). 901

Anemia or Leukemia. 902

Syphilis (1205). 903

Injury. 904

Hydrocephalus (960). 905

Meningitis (590, 608). 906

Cerebral Abscess or Sinus Thrombosis (508). 907

Cerebral Tumor (507, 578). 908

Terminal stage of Optic Neuritis (865). 909

Senile Optic Atrophy. 910

Tabes (827). (Figs. 24-7.) 911

Paresis (1104). 912

Disseminated Sclerosis (668). 913

Disease of the eyeball and orbit (1322). 914

EVERY DAY OF THE YEAR
YOU NEED
**SICKNESS AND ACCIDENT
PROTECTION**

RIDGELY POLICIES PAY
WEEKLY
SICKNESS BENEFITS
WEEKLY
ACCIDENT BENEFITS
ACCIDENTAL
DEATH BENEFITS
A SPECIAL
TRAVELING INDEMNITY
LOSS OF AN EYE, HAND OR FOOT
EVERY DESIRABLE FEATURE
OF SICKNESS AND ACCIDENT
PROTECTION
SMALL QUARTERLY COST
GUARANTEED
NON-ASSESSABLE



CHART XIV e

Abnormalities of Hearing, Taste, and Smell

DIAGNOSTIC ANALYSIS OF SYMPTOMS

DIAGNOSTIC SYMPTOMS AND TESTS				ABSTRACT OF SYMPTOMS		DIAGNOSIS					
808 D I S O R D E R S O F H E A R I N G	820 D E A F N E S S A N A K U S I A (355)	W O R D S A N D S S O U N D S	Usually unilateral. May be bilateral. A permanent symptom.	Bone conduction impaired.	No facial paralysis.	{ Severe paroxysmal vertigo and tinnitus aurium.	Ménière's or Labyrinth disease (650, 685, 1019).	918			
				Bone conduction not impaired.	May be associated with facial paralysis.				{ No vertigo. May be heredity. Locomotor ataxia or disseminated sclerosis may be present.	Atrophy of auditory nerve.	919
			Usually bilateral. Very rarely unilateral, and then only a transitory symptom.	{ Associated with symptoms of lesion of the pons or crura cerebri.		Bilateral lesion of the lemniscus. (Fig. 20.)	922				
				{ Associated with symptoms of lesion of the cerebral cortex. Complete deafness does not always occur in a bilateral lesion of the temporal cortex.				Lesion of the temporal cortex on both sides. (Fig. 15.)	923		
			{ Hysterical symptoms (425). No symptom of organic disease.		Hysterical deafness (1074).	924					
			{ WORDS ONLY. Sensory aphasia (222) is present.				Lesion of left superior temporal convolution. (Fig. 15.)	925			
			821 HYPERAKUSIA, OXYAKOIA OR PARAKUSIA (372, 389).	{ Hysterical symptoms are present.		Hysteria (1074).			926		
				{ Inflammatory lesions of ear or its neighborhood are present.			Hyperemia of inner ear.	927			
				{ Facial paralysis is present. Low notes are especially painful. Tinnitus aurium is present.						Facial paralysis (1317).	928
809 DISORDERS OF SMELL AND TASTE.	Very little, if any, diagnostic significance can be attached to disturbances of smell and taste.										

CHART XV

Perversion of Sensation: Pain and Vertigo

DIAGNOSTIC ANALYSIS OF SYMPTOMS

DISORDERS OF SENSATION—PERVERSION

SYMPTOMS ANALYSED	LOCATION OF PAIN	
930 PERVERSION OF SENSATION IN NERVOUS DISEASES (306).	931 PAIN (330).	933 PAIN IN NERVE Pain limited to the trunk and branches of one nerve in any part of the body, except that at the height of the attack, there may be a mild radiation of the pain into corresponding nerve of opposite side or into adjacent nerves.
		934 PAIN IN HEAD. HEADACHES IN NERVOUS DISEASE After a careful examination with suitable instruments has proved the absence of glaucoma, iritis, muscular insufficiencies and other diseases of the eye, of the nose and its sinuses, of the teeth, of the ear, of the scalp (rheumatism), or of the cranial bones (periostitis, caries).
		935 PAIN IN TRUNK IN NERVOUS DISEASE After a careful examination has proved the absence of Pott's disease, rheumatism of spine or trunk muscles, arthritis, disease of breast, pericarditis, pleurisy, aneurism, pleurodynia, peristitis, cancer and other tumors, colic (intestinal, uterine, biliary, renal) dyspepsia, pancreatitis, appendicitis, peritonitis, gastric ulcer, gastritis, enteritis, hernia, floating kidney, tubal pregnancy, pelvic inflammation, intestinal obstruction, etc.
		936 PAIN IN EXTREMITIES IN NERVOUS DISEASE After a careful examination has proved the absence of any disease of the bones, muscles, joints, blood vessels or skin of the arms and legs. Flat foot must be excluded.
	932 VERTIGO	

See Chart XV a.

See Chart XV b.

See Chart XV c.

See Chart XV d.

CHART XV a
Pain in Nerve, Pain in the Head, Headache

Comprising Numbers 933 and 934 on the left side of Chart
and 937 to 966 on the right margin

DIAGNOSTIC SYMPTOMS AND TESTS

933	PAIN	The differential diagnosis between neuritis and neuralgia cannot always be made clinically. The diagnosis is aided by the experience that certain nerves, such as the sciatic, are more prone to neuritis; while others, such as the trigeminal, are more prone to neuralgia. (Figs. 33, 38).	Paroxysmal pain with free intervals.	Never any motor paralysis or persistent anesthesia or loss of reflexes.	May be of the or ne
			Continuous pain with exacerbations.	May be motor paralysis or anesthesia or loss of reflexes or all combined.	May be distur the el tion.
934	PAIN IN THE HEAD	A history of neurotic heredity or other evidence of a neuropathic predisposition, congenital or acquired, is common. Pain is unilateral and is increased by movement and by exposure to cold or wind, and is sometimes associated with muscle spasm. Vasomotor and trophic disturbances are often present.	Pain limited to the whole or a portion of the trunk and distribution of the trigeminal or occipital nerves. Diseases of the eye, the nose and its sinuses, the teeth, the ear, the scalp and the bones must first be excluded. (For the diagnosis between neuritis and neuralgia see 933.)	The pain is felt above the eye in the forehead, in If tension of eyeball be increased, examine eye.	
			Pain strictly limited to one-half the head.	Periodical attacks (often occurring at menstrual ep crania angio-paralytica) or pallor and dilated p there are vomiting and nausea. The disease u	
			Pain as if nail was being driven through the skull.	Pain of great intensity in a small spot :	
			History or other evidence of syphilis.	Pain may be felt at any time but is worse in eve Cranium is often tender at points. Pain may	
			Disease exists in organs within the head or body.	Frontal headache may be due to gastric dyspepsi to pelvic disease. These referred pains are ass	
			Exogenetic.	Occurs after the ingestion of narcotics. Does not	
			Auto-genetic.	Occurs as the result of breathing for hours foul ai	
			Cerebral hyperemia.	Occurs as the result of constipation, especially wh	
			Cerebral anemia	Occurs in Bright's disease, usually is worse when	
			Evidence of circula- tory disorder.	Headache with fulness and throbbing in head, aches may be followed by a cerebral hemorrhag	
935	PAIN IN THE HEAD	Evidence of nervous exhaustion	Headache associated with phobias and tremors and insomnia and other sy pressure within the skull, especially pressure in occipital and cervical reg	Headache with fulness and throbbing in head, aches may be followed by a cerebral hemorrhag	
			Optic neuritis or choked disc.	Progressive symptoms, motor or sensory or both, cussion over the seat of the lesion. Lumbar pu	
			May follow trauma- tism.	Intractable, incurable, more or less constant head- stretching of the dura mater by tumor, hydroce	
			Evidences of rheuma- tism elsewhere.	Diffuse pain and tenderness of scalp. Pain on m	
			Chronic headache. Pain constant with exacerbations.	Temporary. Occurs during the first few day	
936	PYREXIA	Evidence of infection.	Headache.	Permanent. Occurs throughout the disease	
				Suppuration elsewhere in head	
937	HYPER-PYREXIA	Evidence of exposure to high temperature.	History of exposure to high temperature. Head		

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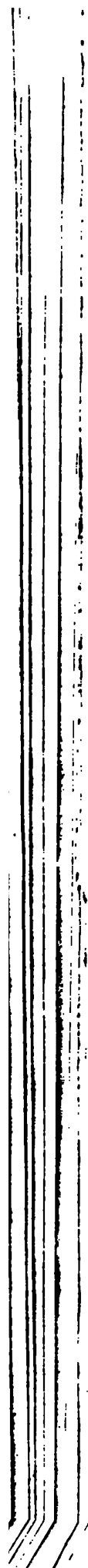


CHART XV b

Pain in Trunk

Comprising Numbers 935 on left side of Chart
and 970 to 990 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

Pain in back.	Evidence of neurotic temperament. No evidence of organic disease.	Pain and tenderness of spinous processes.	{ Phobias and nervous exhaustion, pain and sense of pressure.
	Evidence of organic disease. Pain, tenderness and rigidity of spine.	Pain and tenderness of coccyx.	{ Hysterical symptoms (425). Much tenderness of spine.
Pain in thorax and abdomen.	Girdle pain (374).	May follow traumatism.	{ Severe and constant pain in back and radiating about the trunk. Much spasm of spinal muscles. Exaggerated reflexes if any, it is of a transitory nature. Hyperesthesia.
		Vertebral column is ankylosed.	{ Slowly increasing motor and sensory symptoms, irritative and paralytic symptoms are more prominent, the tumor is large and pain radiating into extremities than in meningitis.
Pain in thorax and abdomen.	Girdle pain (374).	Unilateral.	{ It may be possible to feel exostoses on vertebrae. Unilateral pain in other parts of the body. X-ray examination makes diagnosis.
		Bilateral usually.	{ No other symptoms. Pain shoots around chest, following course of nerve, or may be limited to a small area. Myocarditis, pericarditis, pneumonia, pleurisy have been excluded by a careful examination.
Pain in thorax and abdomen.	Local pain.	At first unilateral and later bilateral.	{ Many other symptoms. Loss of knee-jerk. Argyll-Robertson's pupil.
		In mammary gland.	{ There is a zone of hyperesthesia severe.
Pain in thorax and abdomen.	Local pain.	In precordia and arm.	{ History or other evidence of syphilis.
		Along attachment of diaphragm.	{ No other symptoms. Slowly increasing motor and sensory symptoms.
Pain in thorax and abdomen.	Local pain.	In abdomen. In all these rare forms of neuralgia organic abdominal disease must be carefully and thoroughly excluded.	{ Paroxysmal attacks of pain in or around the chest can be detected. Pain is usually severe.
		In genitals.	{ Paroxysmal attacks of pain in precordial region, of suffocation and imperiousness.
Pain in thorax and abdomen.	Local pain.		{ Pain similar to the above, but not relieved by tobacco, overwork, etc.
			{ Pain felt in lower anterior part of chest, also in same region. Tender points are along the attachment of the diaphragm. Extremely rare disease.
Pain in thorax and abdomen.	Local pain.		{ Paroxysmal attacks of pain in epigastrium often occur in association with each or neighboring viscera, especially no gall stones.
			{ Similar paroxysmal attacks of severe pain, occurring in the abdomen.
Pain in thorax and abdomen.	Local pain.		{ Paroxysmal attacks of severe pain in abdomen occurring in the abdomen. Pain relieved by pressure. Blue line on edge of gum.
			{ Pain in hip, groin, hypogastrium and genitals. Tender points are along the attachment of the diaphragm.
Pain in thorax and abdomen.	Local pain.		{ Neuralgic pains and irritability in the pelvic viscera, tenderness of the testicles, pains at times occur during years in one testicle or

CRITICAL ANALYSIS OF SYMPTOMS

PAIN IN TRUNK

FRACTURE OF SYMPTOMS

in the cervical spine and occiput.

usually in mid-dorsal region; ovarian tenderness is also common.

induced by motion, touch, defecation, etc. In most cases there is a history of injury. Often hysterical

in the extremities.
 { Injury. Very sudden onset. Lumbar puncture may show bloody fluid. Retention of urine.
 { History of infection (septic, syphilis, etc.). Lumbar puncture shows globulin and increase of cellular elements in cerebro-spinal fluid.

paraplegia dolorosa). When irritative symptoms are very prominent the tumor is meningeal, when symptoms at first usually unilateral, later bilateral. Less pain and spasm in back, more girdle pain

girdle pains at level of the disease. Rarely any paralytic symptoms. Usually bone lesions in

intercostal { Tender points of Valleix: one, two inches from posterior median line; another, two inches from anterior median line; and a third, in mid-axillary line. Other points on nerve may also be hyperalgesic. Pain is paroxysmal. Respiration, cough, sneezing, etc., are painful.
 { Rash of herpetic vesicles along course of nerve.

Lumbar puncture gives lymphocytosis. Ataxia. Lightning pains in legs. History of syphi-

sis is and below a bilateral anesthesia, which may be slight, and a motor paralysis, which may be

Lumbar puncture gives lymphocytosis. Pains worse at night.

at first irritative, later paralytic. Brown-Séquard's paralysis at first (442).

and, at times, radiating beyond the limits of the breast. No tumor or other disease of the gland

to left shoulder and even down left arm and, at times, both arms. Sense of oppression in sternal tension is usually high. Area of cardiac dullness usually increased.

neurotic individual who has an overstrained heart. At times the result of gastric indigestion,

frequently on left side. Breathing, sneezing, coughing, etc., painful. Pain occurs in paroxysms. Sternal-cleido-mastoid muscle. No signs of pulmonary, pleural, cardiac or other disease. An ex-

cess, especially in the early morning. No digestive disturbances or evidence of any disease of stomach with contraction of the empty stomach and consequent feeling of hunger.

in the neck of bladder or anus, associated with symptoms of tabes (661).

city, when biliary, renal and other forms of colic, appendicitis, diverticulitis, etc. have been excluded. in urine after administration of K. I.

on crest of ilium, inner part of groin, etc.

uterus, vagina and urethra, but these are rare and relatively unimportant conditions. Neuralgia From this point the pain may radiate.

DIAGNOSIS

Neurasthenia (1072). 970

Hysteria. Spinal Neuralgia (1074). 971

Coccygodynia. 972

Hemorrhachis (524). 973

Meningitis Spinalis, acute (febrile) and chronic (afebrile) (608, 1005). 974

Spinal Tumor (509, 826, 836-40, 981, 1006). (Figs. 24-7.) 975

Spondylitis Deformans. Arthritis Deformans. 976

Intercostal Neuralgia. 977

Herpetic Neuritis (940). 978

Tabes (827, 661). (Fig. 27.) 979

Transverse Myelitis (Figs. 24-7) 980

Syphilitic Meningitis. 980a

Spinal Tumor (975). 981

Mastodynia. 982

Angina-Pectoris. 983

Pseudo-Angina-Pectoris. 984

Phrenic Neuralgia. 985

Gastralgia. 986

Tabetic Crises (433, 827). 987

Enteralgia (Lead Colic, etc.). 988

Lumbo-abdominal Neuralgia. 989

Pelvic Neuralgia. 990



CHART XV_c
Pain in Extremities

Comprising Numbers 936 on left side of Chart
and 995 to 1012 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

DIAGNOSTIC

PA

ABST

Unilateral. Any of these forms of neuritis may be associated with, or precede, or follow a rash of herpes: herpetic neuritis. (Figs. 33, 38).	Pain in arm.	Pain radiates along one or all of the nerves of the arm at other points where nerves are superficial. Vaso-motor paralysis; but movements of arm are impaired by tenderness. Pressure on nerves must be carefully excluded.
	Pain limited to the trunk and distribution of the sciatic, anterior crural or obturator nerve.	Pain shooting along the trunk, or over small areas in the trunk, but the pain may prevent motion. Patient holds the opposite side and bears his weight on the healthy leg. The major (trochanteric point) and in popliteal space may be decided muscular weakness and atrophy. Examination for any possible pressure upon the nerve should be made.
	Pain limited to outer surface of thigh.	Pain along the trunk and distribution of the anterior crural nerve. Tender points on anterior aspect of the thigh. Paralysis and atrophied and knee-jerk lost and anes-thesia may be secondary to diabetes and injury. There may be tenderness.
	Pain in a joint.	Pain along inner side of thigh, along course of obturator foramen. Neuralgia and is usually associated with paralysis of the leg.
	Pain at insertion of Achilles tendon.	Pain is associated with paresthesiae (especially numbness). The paresthesiae are more characteristic of this disease.
	Pain in heel.	Pain in a joint, usually the knee-joint, increased on movement. Absence of any disease of the joint. Many hysterical cases.
	Pain in toe.	Severe pain at insertion of Achilles' tendon on walking.
	With girdle pains, and lumbar puncture gives lymphocytosis.	Pain in lower surface of heel, especially when walking. Removal of the sub-calcaneal bursa, or of exostoses.
	With anesthesia.	Pain in the metatarso-phalangeal joint, especially of the first toe. The arch is lowered from "breaking" of the arch transverse.
	With dissociation of sensation.	With Romberg's symptom, Argyll-Robertson's phenomenon, lymphocytosis and lightning pains over small areas.
Bilateral.	With vaso-motor disturbances.	With pain and rigidity in back and in extremities.
	With fat.	Steadily progressive motor and sensory symptoms, as in Brown-Sequard's paralysis (442).
		Motor paralysis and anesthesia over whole of both legs. Peripheral and organic reflexes. Muscular atrophy in lower back and radiating into legs.

ANALYSIS OF SYMPTOMS

EXTREMITIES

OF SYMPTOMS

der points in supra-clavicular fossa, in axilla at head of radius and at
 disturbances. Fibrillary contractions at times occur. There is no motor
 Tumors at base of neck and in axilla, and a cervical rib (556), causing

uation of the sciatic nerve. Little, if any, anesthesia or motor paralysis,
 ne affected side semi-flexed, thigh slightly abducted, inclines his body to
 tender points over the sciatic notch (gluteal point), above the trochanter
 point). In neuritis, the nerve, wherever felt, is tender, and then there
 is much more frequently a neuritis than a neuralgia. A rectal examina-
 tion should be made.

erve on the anterior surface of the thigh and inner surface of leg to the
 inner side of knee and at internal malleolus. Extensors of thigh may be
 be on anterior surface of thigh and inner side of leg in neuritis. May
 ition of herpes along the course of the nerve.

after hernia and other diseases have been excluded. A rare form of
 factors.

tingling) and is frequently associated with, and is caused by, flat foot.
 is the pain, which is often entirely absent.

The skin is much more sensitive than the articular surfaces. No evi-
 dence (425).

ending. May follow gonorrhea, malaria, gout, broken arches or injury.

ling. Some cases are cured by rheumatic medicine, others by surgical
 y supporting the weakened arches.

toe, usually following an injury. Usually occurs in women. The joint

axia, history of syphilis usually, always loss of knee-jerk, cerebro-spinal
 superficial and deep, often followed by hyperalgesia over same area.

ed reflexes. No ataxia. No Argyll-Robertson's phenomenon.

ly unilateral, later bilateral. Increased pressure of cerebro-spinal fluid.

in some cases the domain of the anterior crural nerves. Abolition of
 tic disturbances. Anesthesia in perineum and genitals and much pain

Extremities and extending towards body. Muscular weakness, atrophy
 Paresthesiae in toes and fingers and often with fever.

at tactile anesthesia. Trophic disturbances and mutilations. These
 tic paraplegia in legs. The pains often resemble the pains of tabes, but

and later with pallor, shrinking and wrinkling of the same parts. Flat

l congestion; so that fingers and toes become purplish and even black.
 off.

arms and legs, but not elsewhere. There is considerable pain associated
 stages when they are forming.

DIAGNOSIS

Cervico-brachial Neuralgia or Neuritis of Ulnar, 995
 Median Radial, etc.

Sciatica (720). 996

Crural Neuralgia or Neuritis. 997

Obturator Neuralgia. 998

Meralgia Paresthetica. 999

Arthralgia or Hysterical Joint. 1000

Achillodynia. 1001

Talagia or Calcanodynia. 1002

Metatarsalgia or Morton's Toe. 1003

Tabes. Neuralgic stage (661). 1004

Spinal Meningitis (608, 974). 1005

Spinal Tumor (509, 826, 836-40, 975). 1006

Lesions of Cauda Equina (487). (Fig. 29.) 1007

Multiple Neuritis (488). 1008

Syringomyelia (552, 693, 837, 1150a, 1170, 1187, 1009
 1357-9).

Erythromelalgia (1198). 1010

Raynaud's Disease (1195). 1011

Adiposis Dolorosa. Dercum's Disease (1176). 1012

No.	Name	Address
1	John Smith	123 Main St, New York, NY
2	Jane Doe	456 Elm St, New York, NY
3	Robert Brown	789 Oak St, New York, NY
4	Mary White	101 Pine St, New York, NY
5	James Green	202 Cedar St, New York, NY
6	Elizabeth Black	303 Birch St, New York, NY
7	William Gray	404 Spruce St, New York, NY
8	Anna King	505 Willow St, New York, NY
9	Thomas Lee	606 Ash St, New York, NY
10	Sarah Hall	707 Hickory St, New York, NY
11	Charles Adams	808 Sycamore St, New York, NY
12	Patricia Baker	909 Magnolia St, New York, NY
13	Richard Wilson	1010 Poplar St, New York, NY
14	Laura Miller	1111 Dogwood St, New York, NY
15	George Taylor	1212 Redwood St, New York, NY
16	Helen Moore	1313 Cypress St, New York, NY
17	Frank Johnson	1414 Juniper St, New York, NY
18	Grace Harris	1515 Fir St, New York, NY
19	Edward Clark	1616 Hemlock St, New York, NY
20	Frances Lewis	1717 Larch St, New York, NY
21	Harold Walker	1818 Alder St, New York, NY
22	Martha Young	1919 Hawthorn St, New York, NY
23	Benjamin King	2020 Laurel St, New York, NY
24	Julia Scott	2121 Rose St, New York, NY
25	Samuel Green	2222 Violet St, New York, NY
26	Elizabeth Adams	2323 Iris St, New York, NY
27	William Baker	2424 Dandelion St, New York, NY
28	Anna Miller	2525 Pansy St, New York, NY
29	Thomas Wilson	2626 Tulip St, New York, NY
30	Sarah Taylor	2727 Marigold St, New York, NY
31	Charles Moore	2828 Zinnia St, New York, NY
32	Patricia Clark	2929 Begonia St, New York, NY
33	Richard Lewis	3030 Petunia St, New York, NY
34	Laura Walker	3131 Hyacinth St, New York, NY
35	George Young	3232 Lavender St, New York, NY
36	Helen King	3333 Geranium St, New York, NY
37	Frank Scott	3434 Snapdragon St, New York, NY
38	Grace Adams	3535 Fuchsia St, New York, NY
39	Edward Baker	3636 Camellia St, New York, NY
40	Frances Miller	3737 Hibiscus St, New York, NY
41	Harold Wilson	3838 Peony St, New York, NY
42	Martha Taylor	3939 Aster St, New York, NY
43	Benjamin Moore	4040 Gladiolus St, New York, NY
44	Julia Clark	4141 Iris St, New York, NY
45	Samuel Lewis	4242 Pansy St, New York, NY
46	Elizabeth Walker	4343 Marigold St, New York, NY
47	William Young	4444 Zinnia St, New York, NY
48	Anna King	4545 Begonia St, New York, NY
49	Thomas Scott	4646 Petunia St, New York, NY
50	Sarah Adams	4747 Hyacinth St, New York, NY

CHART XV d

Vertigo

Comprising Numbers 932 on left side of Chart
and 1015 to 1033 on right margin

1. The first of these is the fact that the number of cases of the disease has increased in the last few years. This is due to a number of causes, the most important of which are the following:
2. The second is the fact that the disease is now more common in the lower classes of society. This is due to the fact that the lower classes are more exposed to the disease than the upper classes.
3. The third is the fact that the disease is now more common in the cities than in the country. This is due to the fact that the cities are more crowded and the people are more exposed to the disease.
4. The fourth is the fact that the disease is now more common in the winter than in the summer. This is due to the fact that the winter is a more favorable season for the disease.
5. The fifth is the fact that the disease is now more common in the south than in the north. This is due to the fact that the south is a more favorable climate for the disease.
6. The sixth is the fact that the disease is now more common in the east than in the west. This is due to the fact that the east is a more favorable climate for the disease.
7. The seventh is the fact that the disease is now more common in the south-east than in the north-west. This is due to the fact that the south-east is a more favorable climate for the disease.
8. The eighth is the fact that the disease is now more common in the south-west than in the north-east. This is due to the fact that the south-west is a more favorable climate for the disease.
9. The ninth is the fact that the disease is now more common in the south-east than in the north-west. This is due to the fact that the south-east is a more favorable climate for the disease.
10. The tenth is the fact that the disease is now more common in the south-west than in the north-east. This is due to the fact that the south-west is a more favorable climate for the disease.

CHART XVI
Disorders of Cerebral Activity

DIAGNOSTIC ANALYSIS OF SYMPTOMS		
SYMPTOMS ANALYSED	ALTERATIONS IN MENTALITY	
1036 Disordered Mentality.	1037 Coma.	See Chart XVI a.
	1038 Pseudo-Coma.	} See Chart XVI b.
	1039 Double Personality.	
	1040 Weakened Mentality.	
	1041 Insanity.	See Chart XVI c.

CHART XVI a

Coma

Comprising Numbers 1037 on left side of Chart
and 1042 to 1068 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

303;
COMA OR
SEMI-COMA
(205, 745).

See also
pseudo-coma,
(1038).

History, or other evidence, of recent injury to head.	Convulsions rare.	<p>Pupils usually contracted and respond feebly to light. Patient may be completely unconscious; symptoms follow the injury immediately.</p> <p>Pupils dilated, often unequal, and usually do not respond to light. Symptoms usually follow the injury; symptoms. Often conjugate deviation.</p>
History of a previous brain illness, of which the coma is only one symptom, and often the terminal one, or the presence of an inflammation of the scalp (erysipelas, suppuration), or of the bones of the skull (caries and especially suppuration of the bones of the ear).	Convulsions are frequently present.	<p>The symptoms are those of a local meningitis (507, 1045) or abscess; the coma are common. There may be local symptoms, both irritative and paralytic, such as be edema of eyelids and conjunctivae, choked disc, prominence of the optic disc.</p> <p>Retraction of neck and opisthotonus. Fever, headache, delirium. Convulsions followed by those of paralysis.</p> <p>Headache, vertigo and vomiting. Often mild delirium. A recent, in general and resemble those of a rapidly growing tumor. Choked disc.</p> <p>Headache, vertigo and vomiting. May be a history of former injury; tumors at the base are more likely to cause paralysis of one or more limbs.</p> <p>A motor monoplegia (anarthria, etc.), rare, hemiplegia or diplegia or paraplegia. Epileptiform convulsions, unilateral or bilateral, are frequent.</p> <p>Progressive mental impairment, childishness, restlessness, amiable but incoherent; syllables and letters are left out and letters doubled. Apoplectiform.</p> <p>History of lead poisoning, of lead colic, of wrist-drop, etc. Blue line on gums.</p> <p>Intention tremor. Scanning speech. Many motor and sensory symptoms.</p>
History or other evidence of poisoning.	Convulsions rare.	Headache, increasing fever, difficulty in walk and speech, tremor, incontinence.
	Convulsions absent.	<p>Patient can usually be aroused from his coma sufficiently to speak and give history of alcoholic abuse.</p> <p>Pupils are contracted and do not respond to light (if opium). Patient may be completely unconscious; symptoms follow the injury immediately.</p> <p>Patient is in a confined space or room in which there is a strong smell of gas.</p>
Evidence of a cardiac inadequacy.	Often a slight spasm or rigidity during the attack.	<p>Sudden attack of coma with pallor and weak or absent cardiac action; extremities cold, restlessness, yawning. Low arterial tension, steady.</p> <p>Sudden intermission of heart beat during a considerable fraction of the attack.</p>
Sudden attack of coma of short duration with or without a convulsion. Usually a history of similar attacks and often of remote injury.	Convulsions almost always present and are usually the most striking symptom of the disease, but not so characteristic as unconsciousness, which is at times the only symptom of the attack.	<p>The attack usually commences with a convulsion, as described in Charcot's book; sometimes trivial, sometimes a deed of violence (post-epileptic insanity); unconsciousness and no convulsion and either no action or some trivial action. The convulsive attack and is called the "psychic equivalent" and is followed by a period of unconsciousness and take journeys and are lost to their friends and to themselves. An epileptic attack is the complete or almost complete unconsciousness and some mental weakness which may slowly increase to mild or extreme. Epileptics have attacks during many years and yet show little, or no, change.</p> <p>The attack is altogether similar to the major attack of epilepsy, but is followed by a distended abdomen, foul smelling feces, vomiting, diarrhoea, etc., and a period of unconsciousness.</p>
Sudden attack of unilateral paralysis. Rarely the paralysis comes on slowly; steadily increasing during hours or days, "Ingravescent apoplexy." In such cases the coma may be slight or absent.	Convulsions are rare, but both tonic and clonic spasms may occur, involving one-half of the body when the lesion is cortical, or involving both sides of the body when the hemorrhage occurs in the brain stem (460) or ruptures into a ventricle.	<p>The coma comes on instantly or in the course of a few hours. There are no convulsions. The bilaterally innervated muscles (upper extremities, abdominal, micturition, defecation, etc.) escape permanent paralysis; reflexes may, during the coma, be abolished, later exaggerated (Reflexes pass away after several hours or days. The lower branch only of the paralyzed side. Usually there is also at first a sensory hemiplegia in which latter case the motor paralysis greatly improves or entirely disappears in leg and finally in the arm. In case recovery does not take place in leg. Post hemiplegic motor disturbances may occur, especially in the arm. Some motor weakness may occur. A reactive inflammation about the second week. Repeated attacks at varying intervals are common.</p>
Albumen and casts, or sugar, or all three, in urine.	No convulsions.	Patient emaciated. Acetone odor of breath. Pulse is small and rapid.
	Convulsions usually.	Onset usually gradual. Some edema, cyanosis, restlessness, rapid noise.
Decided fever.	Convulsions frequently.	Occurs at the onset of acute infections, especially in children. Often followed by a period of unconsciousness but in these cases convulsions are rare, and the cause may consist in a local infection.
Hyperpyrexia.		History or evidence of exposure to great heat. Absence of perspiratory symptoms (paralytic) occasionally occur.

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CHART XVIb

Pseudo-coma, Double Personality and Weakened Mentality

Comprising Numbers 1038 to 1040 on left side of Chart
and 1069 to 1075 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

1425
PSEUDO-COMA.

Hysterical symptoms (425).

Convulsions and Spasms frequent.

Occurs usually in girls and women of a nervous type. Can be stopped by a strong and continued suggestion. Will usually show that she is conscious of her condition from external causes, or auto-suggestion hypnosis.

1426
DOUBLE PERSONALITY AND AUTOMATISM (298).

Hysterical symptoms (425).

Convulsions frequent.

Patient seems at times to be in a hypnotic state, and in that state to lead a life earned a brain states from auto-suggestion, patients often exhibit and in some hysterical patients may exhibit.

Epileptic symptoms (425).

While in an unconscious state patient often has no memory. Whether in such unconscious state is doubtful. While unconscious, epilepsy often occurs.

Apprehension and various phobias are prominent symptoms. In consequence of their weakened mentality, these patients cannot rid themselves, by reasoning, of their unreasonable apprehensions and fears.

The symptoms are those of a general debility, especially of the lower centers. It is more easily. Every task becomes as a mental labor will power are both poor. They feel nervous, phobia, claustrophobia, etc.). About a chorea. The patient also suffers much from palpitation, digestive disturbances. The essential symptom is it.

The patient, usually a female, is in a condition of consciousness in which the attention is fully expressed because of a delusion in regard to a false idea, but the false idea cannot be dispelled but is strong, fantastic and impossible. At one time and apprehensive, and their attention is fully even grotesque delusion, that some organ is dead.

Abnormal and greatly increased suggestibility is the prominent symptom. Symptoms are varying, inexplicable and incredible. No certain evidence of any organic disease, although almost every disease can be more or less perfectly simulated (425).

The disease occurs almost exclusively in women of a nervous system, are probably all really cerebral and not adopted by the patient as the result of organic patients are usually so dominated by the desire of accomplishing this. Too much value and stimuli varies from day to day and is often (425). Anesthesia, parosmia, hysterical disturbances occur alone or combined, hysterical often has as its cause a psychic trauma, like a nervousness, theatrical posing, irritability and sense of remorse and starting episodes of life. In general these patients show lack of self may show wonderful will power or desire. "Trance of hysteria," others occur only rarely. The cataplexy (425), globus hystericus (35), esophageal, torticollis and other spasms (38), (42), ovarian tenderness, photophobia, vertigo, incontinence and feeling, tremor, flat fever, flushing, sweating, angio-neurotic edema, field of vision, (388) somnambulism (388), etc.

1440
WEAKENED MENTALITY. Patients appear to be intelligent, but incapable of long sustained effort, and of self-control, often foolish and unreasonable. The different diseases in these groups merge into each other and no sharp line can be drawn between them. They all rest on a neurotic basis, and in all suggestion plays a great part.

The result of an accident.

The disease occurs as the result of trauma or injury. It very rarely occurs when a woman has been when pecuniary compensation may be obtained by any compensation. The disease is described above under hysteria. Trance, falls, paralysis (motor and sensory), palpitation and are insomnia, especially in the early morning symptoms can be simulated, and as may be seen and unconscious simulation. Suggestibility in "suggestion," as in hysteria.

All the various forms of insanity described in the next chart exhibit, and are in part dependent

S OF SYMPTOMS

7, AND WEAKENED MENTALITY

SYMPTOMS

Eyelids are closed and resist attempts to open them. Coma can usually be induced. Even in the apparent coma the patient is suggestible and close observations and therefore not truly comatose. Such a condition may result in somnambulism, trance).

Allied condition from auto- or foreign suggestion, or from wilful deception, similar states quite distinct from the normal life. In the hypnotic or allied states. This is a very rare condition and offers much opportunity for deception—unconscious suggestion on the part of the physician.

Complicated acts and leads a life, during hours, days or weeks, of which he later can remember what happened in previous similar states is, to say the least, automatic acts.

Nervous system, especially of the brain, associated with an increased irritability, but more so in women. The patients are either incapable of exertion or tire so that they are discouraged before they undertake it. Their memory and judgment are impaired and have a number of peculiar fears: phobias (235—agoraphobia of neurasthenia as are the phobias, are indecision and lack of will power. Motor disturbances, paresthesiae, headache, backache, neuralgias and digests—neurasthenia are apprehension and fear (phobias).

Neurasthenia and is greatly depressed by reason of an abnormal state of self—permanently fixed upon the condition of his body or of his mind. Patient is depressed or abnormality of some organ of his body, generally the viscera, which on physical examination no abnormality can be discovered adequate to justify the patient's mind. These false judgments are very various and are often monotonous an exaggeration of the neurasthenic phobias. The patients are anxious about their ills. The essential symptom of hypochondriasis is a fixed, constant, morbidly diseased.

Imagination, and the symptoms, which may apparently affect any part of the nervous system: to be the result of a false idea (delusion—215), or of suggestions derived from others or from some abnormal sensations within the body. The patient's wonder and admiration that they are not very scrupulous in their means, more, be placed on their statements. The reaction of the patients to external influences in its results. The symptoms of the disease are both many and variable—paralyses, convulsions, spasms, contractures, vaso-motor and secretory changes, producing a confused and constantly varying picture of disease, which is chronic, or more frequently both. In addition to the chronic condition of suggestibility, the course of the disease is interrupted by the sudden appearance of great intensity, which render the patient helpless and often apparently threaten death, but in the production and maintenance of some prominent symptom they these symptoms occur so frequently that they have been called the "stigmata" of these acute hysterical attacks are convulsion (586), coma (1069), loss of laughing or crying, aphonia (748, 759), mutism (747), stricture of sensation and its transference (425, 834), astasia, abasia (653, 792), paralysis of anal irritation, clonus (950), cough, dyspnoea, palpitation, vomiting, regurgitation, false pregnancy, peritonitis, anuria, polyuria, melanuria, hemorrhages, blindness (851a), deafness, (924), anosmia, ageusia, concentric limitation of consciousness (1039), etc.

with great fright, or in some accidents from fright alone without physical injury has been received. It is especially common in railroad accidents and in cases of the injury; although it occurs also in cases where there is no hope of recovery to neurasthenia and hysteria and it may present any of the symptoms mentioned, especially after exertion, vertigo, paresthesiae, neuralgic pains, local motor disturbances are common symptoms. Quite characteristic of the disease is a melancholic, hypochondriacal, mental state. Most, if not all, of these patients are seeking to recover damages, there is naturally more or less of contentment, is far from explaining the traumatic neuroses, the key to which lies rather

in a weakness of the mental powers, varying in degree, but always decided.

DIAGNOSIS

Hysterical Coma (1074).	106
Hysteria (1074).	107
Epilepsy (1058).	107
Neurasthenia, Psychasthenia (113, 155, 161, 163, 178, 180, 671, 674, 843, 845, 959, 970, 1033).	107
Hypochondriasis (216).	107
Hysteria (111, 128, 130, 153, 179, 345, 425-6, 527, 586, 618, 628, 664, 674, 747-8, 759, 793, 834, 843, 848, 866, 878, 924, 926, 950, 971, 1000, 1033, 1069-70, 1075).	107
Traumatic Neuroses. Sometimes called Traumatic Hysteria (156, 616, 674, 1033).	107



CHART XVIc

Insanity

Comprising Numbers 1041 on left side of Chart
and 1076 to 1117 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

E X T R E M E D E F E C T I N I N T E L L I G E N C E	1076 Amentia (211). More or less complete	A condition in which the mind has not developed with advancing age, due to a disease of the brain, either congenital or acquired in infancy. Besides the mental defect, these patients often present many and various physical defects and deformities such as: deformed skull, posterior hydrocephalus, high palatine arch, coarse body, deformed ears, etc. The amentia may be either general or partial, and some of its slighter degrees may be due in part to defective training.		Patients show little or no intelligence. A urine and feces. About their only desire. Most of these patients exhibit frequent
			Occurring in youth, at puberty or before 25 or 30 years.	Patients can talk and are more or less clever. They are incapable of much education, and the sexual instinct is often strongly
				Patients show a degree of intelligence as children develop up to a certain point, intelligent eyes, thick fissured tongue, and short tobacco. Simple infantilism is often
				Certain feeble-minded persons seem incapable
I N S A N I T Y	1077 Dementia (212) More or less complete.	A condition in which the mind has developed to a certain, even a high, degree of intelligence and then, in consequence of disease of the brain (functional or organic), all mental development has not only ceased, but there has been a distinct retrogression, which may go on to a complete loss of intelligence. Memory, emotions and interest are all lost. Patient becomes apathetic, reacts to no stimulation, soils himself and does not even eat.	Usually occurring in adult life after 25 years, but may occur in youth.	Complete apathy, coming on more or less suddenly. Appears to be anesthetic and in some cases recover after several months.
			Occurring in old age.	Partial apathy. Patients are dull and stupid. There is an absence of emotions and of interest. They perform frequently spontaneous, intelligent acts of a phrase which they have just heard. The varieties under this head merge into
				History of alcoholism and usually associated with
				History of alcoholism extending over many years and large amounts of alcohol. The symptoms at
I N S A N I T Y	1078 Hallucinations are abundant and dominant. Hallucinatory Insanity (213).	A condition in which the patient is constantly receiving false perceptions from his different senses: either visual, auditory, olfactory, gustatory, tactile or painful, or from several or all combined. Associated with this is always a certain degree of impairment of consciousness, which weakens his judgment and does not permit him to decide that these hallucinations are false.		History of very numerous epileptic seizures
				History of a previous psychosis which has been (apathetic dementia) but some cases show
				History of syphilis. Lumbar puncture shows inability for continuous mental concentration at least cheerfulness, in spite of the illness. dementia. No paralysis, but much paresis, tabes, more rarely with various forms of
				Associated with physical weakness and with loss of memory, especially for recent events. Blood tension often high.
I N S A N I T Y	1079 Delusions are present and dominant. Delusional Insanity (215)	A condition in which the patient has formed a false judgment about things which concern him. The basis of these false judgments is partly a congenitally defective brain and partly hallucinations. Associated with these delusions there is always present a varying degree of impairment of intelligence, which prevents the patient from recognizing the falseness of the delusion when evidence is presented to him which would be adequate for a normal man; although many of these patients in their own way reason shrewdly. These delusions lead to irrational conduct on the part of the patient which would not be irrational were the delusions true.		Patient is overwhelmed by the intentions towards him.
				History of alcoholism. Patient is very nervous. Great fright. Violent
				Many other poisons besides alcohol produce a mild hallucinatory insanity
				Disease commences with
I N S A N I T Y	1080 An exaggerated emotional state is the dominant symptom. Emotional Insanity (204). The insanities of the neuroses have been considered under epilepsy, hysteria and hypochondriasis.	Exaggeration of the sometimes natural feeling of sadness or discouragement with life. Exaggeration of the natural feeling of joyousness. Alternations of mania and melancholia.		History of alcoholism. Patient is very nervous. Men is that their wives
				Patient has delusions upon which his life is not built upon them.
				Patient has a number of systematized or nearly systematized delusions which are strong enough to influence conduct and bearing. Curable
				Patient has many delusions which are woven in with the systematized delusions and are thus systematized himself as a most remarkable delusion of persecution
I N S A N I T Y				Patient is constantly in a depressed and pessimistic mood. He is unable to explain the melancholia and is very prone to suicide. Their circles
				Patients are constantly in a joyous and exuberant mood due to delusions (1111). Many
				Alternations at long intervals of mania and melancholia
				Attacks of excitement or of depression may be observed. Kraepelin has incorporated all of these forms





CHART XVII

Trophic and Sympathetic Disorders

DIAGNOSTIC ANALYSIS OF SYMPTOMS

TROPHIC DISORDERS AND DISORDERS OF THE SYMPATHETIC SYSTEM

SYMPTOMS ANALYSED	TISSUES INVOLVED	
1120 Trophic Lesions.	1122 Muscular Tissue.	See Chart XVII a.
	1123 Cutaneous and Sub-Cutaneous Tissue.	See Chart XVII b.
	1124 Fatty Tissue.	} See Chart XVII c.
	1125 Bone Tissue.	
	1126 Joint Disease.	
	1127 Other Trophic Lesions.	
1121 Disorders of the Sympathetic System.	1128 Ganglionic Disorders.	} See Chart XVII d.
	1129 Vaso-Motor Disorders.	

CHART XVII a

Muscular Atrophy and Hypertrophy

Comprising Numbers 1122, 1130 and 1131 on left side of Chart
and 1146 to 1156 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

1122 MUSCULAR TISSUE	1130 ATROPHY.	{ Atrophy is great in degree and rela- tively rapid in onset.	{ Muscular atrophy. Lesion of peri- pheral motor- neurons.	{ Acute and sub- acute course, (inflammatory lesions). Chronic course, (degenerative lesions).
	1131 HYPERTROPHY	{ Atrophy is slight in degree and very slow of onset.	{ Muscular atrophy and hypertrophy combined. Lesion in muscles. Lesion in central motor-neurons.	{ Associated with chr neuritis can be fo Muscles of face (Lan- form) are first affe atrophied, a few h Very slow course.
		{ Increased strength.	No lesion.	Muscular fibers normal. A true hypertrophy.
		{ Decreased strength.	Lesion in muscles.	Calf muscles, infra- trophy. Other m generation; some atrophied. Legs s

IS OF SYMPTOMS

ND HYPERTROPHY

SYMPTOMS

ori- nd on-	Complete or partial electrical reaction of degeneration.	History of injury, wound, bruise or scar.	Injury of nerve (489, 822).	1146
		Limited to distribution of one nerve (simple neuritis) or many nerves, (multiple neuritis). Usually associated with sensory symptoms: pain and anesthesia, nerve and muscle tenderness.	Neuritis (488-92, 822, 940-8). (Figs. 33, 38.)	1147
		Groups of muscles attacked not corresponding to the distribution of any nerve. No sensory symptoms, except some pain at onset in back, joints and muscles. Very rarely nerve and muscle tenderness. Globulin and lymphocytosis in cerebro-spinal fluid in acute stage.	Acute anterior poliomyelitis (495, 789). (Figs. 26-7.)	1148
		Atrophy affects either the arms or the legs. Sensory and other symptoms of myelitis are present. Organic reflexes are more or less disordered. Superficial and deep reflexes are abolished in the paralyzed area.	Myelitis of Cervical or Lumbar Enlargement (485, 549).	1148a
ori- nd nd	Diminution of the electrical excitability, but no reaction of degeneration.	Atrophy commences in the small muscles of hands, or muscles of shoulder girdle, and extends and is associated with fibrillary contractions. Mild spastic paraplegia (525, 797) in legs.	Amyotrophic lateral sclerosis (547, 695, 797). (Figs. 26-7.)	1149
		Atrophy affects the muscles of tongue and lips and is associated with fibrillary contractions. Mild spastic paraplegia (525, 797) in legs.	Chronic bulbar paralysis (546, 694). (Figs. 21-2.)	1150
		Atrophy affects the hands usually. Is associated with dissociation of sensation and often with ulceration and mutilation.	Syringomyelia (552, 693, 837-9, 1009, 1170, 1357-9).	1150a
especially with ankylosis.		Many of these cases are neuritic, but in some no	Arthritic atrophy.	1151
pe), or of shoulder girdle (Erb's juvenile type), or of legs (pseudo-hypertrophic les apparently hypertrophied. Excised muscle fibers show degeneration: some increase of interstitial fat. No fibrillary contractions.			Muscular dystrophies (477, 786, 1156).	1152
phy	The atrophy is due entirely to disuse. No electrical reaction of degeneration.	The reflexes are exaggerated. Ankle-clonus and Babinski are present when legs are affected, unless prevented by contractures.	A paralysis of long standing, especially one from infancy.	1153
is the result of much exercise.			Strong man or athlete.	1154
is due to muscle spasm, occurring at the commencement of voluntary motion.			Thomsen's disease (613).	1155
nd some other muscles appear large, but are weak: a false or apparent hyperak and atrophied. No fibrillary contraction. Excised muscle fibers show deypertrophied and much interstitial fat. Slow course. All muscles are finally ly affected.			Pseudo-hypertrophic paralysis (500) and the muscular dystrophies (1152).	1156

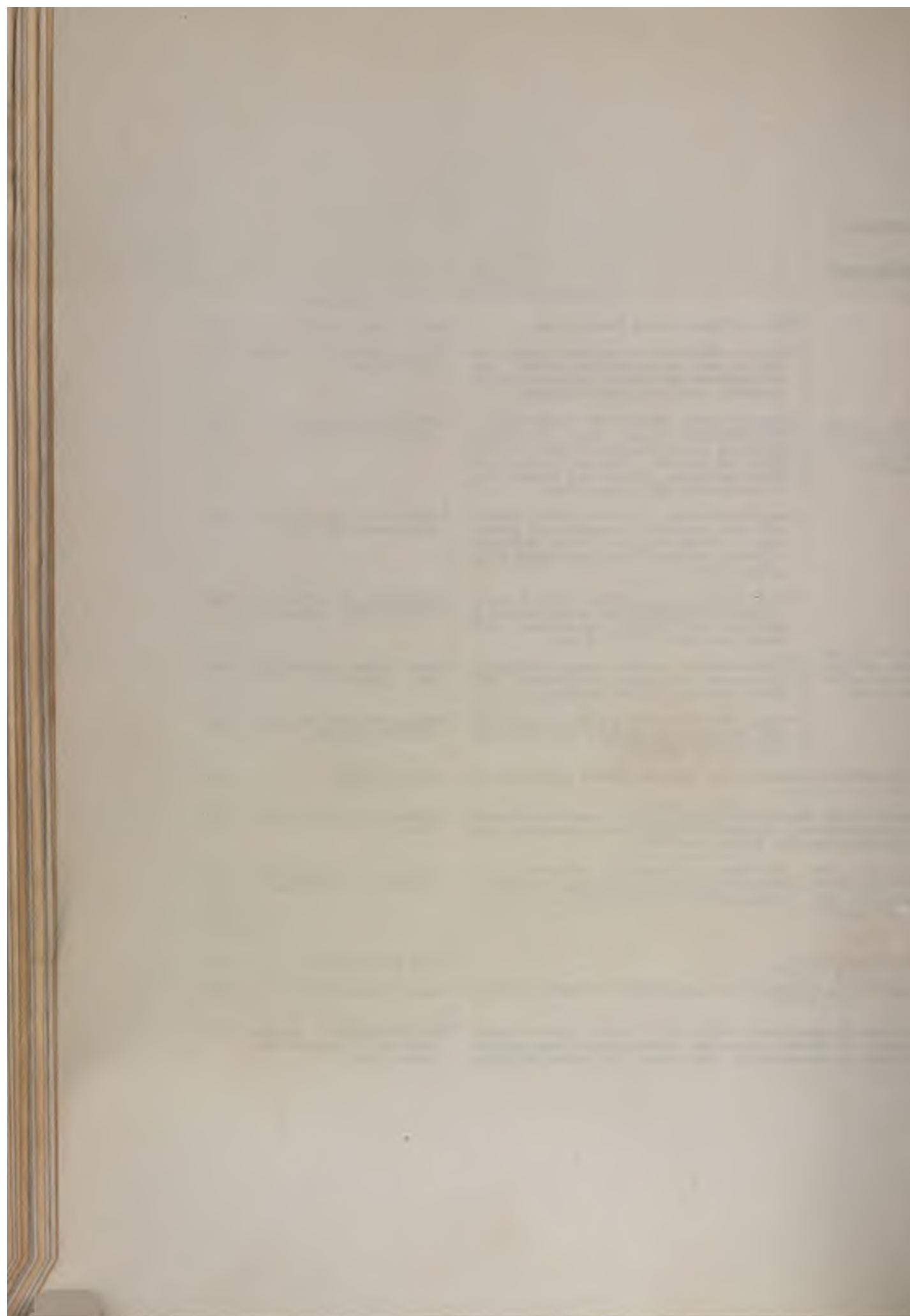


CHART XVII b
Cutaneous and Sub-cutaneous Trophic Disorders

Comprising Numbers 1123 and 1132 to 1135 on left side of Chart
and 1160 to 1173 on right margin

CUTANEOUS AND

DIAGNOSTIC SYMPTOMS AND TESTS

1123
CUTANEOUS
AND SUB-
CUTANEOUS
TISSUE1132
Atrophy.

The skin is unusually smooth and thin. The fingers become thin. This change occurs quite frequently in nervous diseases.

The hair falls out, either all over head, face and body, or the skin is not changed in appearance. Allied to this is the consequence of severe pain, or psychic shock, or

Atrophy of the normal pigment of the skin; so that persons of dark complexion. The edge of the patch is irregular. See also facial hemi-atrophy, 1179.

1133
Hypertrophy.

The skin and mucous membranes everywhere appear thickened, on pressure. The skin is sallow, dry and cracked. The body and features are enlarged. Nails, teeth and hair are heavy. Voice is slow and hoarse. Response is slow. The thyroid gland is atrophied, or removed. Arterio-sclerosis is common in women than in men, and frequently occurs in children they become dwarfs. The cause of the disease is the thyroid gland and it can be cured by the administration of iodine.

The skin is thickened, generally or locally, infiltrated, especially at their ends, and the fingers become swollen. It is more common in women than in men and seems to be allied to myxedema (oedematosum) and ends with an atrophy of the skin.

1134
Eruptions.

Clusters of vesicles filled with clear fluid, each cluster is on one or two nerve roots and strictly limited to their distribution. It is usually accompanied, preceded and followed by pain. The pain may continue for months after the rash has disappeared.

In some forms of nervous disease (especially in hysteria) the skin is irritated (urticaria) and sometimes do not.

Successive crops of bullae, which are at first small vesicles. Several vesicles may coalesce. There may or may not be pain. A very fatal disease.

1135
Ulcerations.

With much loss of tissue.

Ulcerations larger and smaller, with sloughing and loss of phalanges, even whole fingers and toes. The whole process is painless and may in part be the result of trauma in the analgesic parts.

Large, deep, sloughing ulcerations. Patients usually suffering from diabetes, subjected to much pressure. The ulcerations are usually clean.

With small loss of tissue. See also Raynaud's disease (1195).

An ulceration usually commencing on the dorsum of the foot and extending to the toes. Such an ulceration which ulcerates and the patient has laryngeal reflexes and other symptoms in the urine in a small amount.

Ulcerations more or less severe. The skin is often bronzed. Syphilis.

IS OF SYMPTOMS

TROPHIC DISORDERS

SYMPTOMS

The nails are excessively curved and are striated. This is in which the peripheral neurons are degenerated.

Only in patches, usually on the head and face. The hair turning white of the hair in patches, or universally, in (of hair dye).

They appear. They are, of course, most noticeable in the pigmented than the surrounding skin.

ated, and do not pit, or pit but very sensitive to cold. The II out. The movements are y very sluggish and at times ie. The disease may follow is may be present. Is more e climacteric. When it occurs sence of the secretion of the gland.

Occurring in adults.
Occurring in children.

nd. The bones of the phalanges become absorbed, bnormally movable. The disease is more common often commences as a local patch of edema (stadium um atrophicum). At times patches are pigmented.

dened skin; the clusters following the course of one tion dries up and disappears after a week or two. aerve, along the course of which it is situated. The

white or red, appear, at times spontaneously, and 0). Such patches of urticaria sometimes itch and

ny size, appear on the skin and mucous membranes. are always some burning sensations and the pain

ymptoms in legs. The disturbances are limited to distribution of one or more nerves. All forms of are abolished. Small tumors may occur along the es, together with other manifestations of leprosy.

ptoms in legs, when, as is usual, the trophic dis- are limited to hands and arms. Pain and tempera- lost, with persistence of tactile sensibility, usually ed area. Kyphosis and spondylitis are common

ness of the skin and occurring only in bed-ridden paralysis, and occurring almost always on parts etc.), especially when the parts are not kept scru-

se foot, not growing larger superficially, but slowly se; it extends quite through the foot and appears s on the hand. It usually commences as a corn, us. Loss of knee-jerk, Argyll-Robertson's pupil- ent in the majority of cases, while sugar is present

t traumatism. In cases of arsenical neuritis, the are present.

DIAGNOSIS

Glossy skin. 1160

Alopecia, (general or areata). 1161

Vitiligo and Leucoderma. 1162

Myxedema. 1163

Cretinism and Dwarfs (1090, 1177). 1164

Scleroderma and Sclerodactyly. 1165

Herpes Zoster. Herpetic Neuritis. 1166

Urticaria (1201). 1167

Pemphigus. 1168

Leprous Neuritis. 1169

Syringomyelia or Morvan's disease (552, 693, 1187). (Figs. 25-7.) 1170

Bed Sores. Decubitus. 1171

Perforating Ulcer of Tabes and (rarely) Syringo- myelia and Diabetes. 1172

Neuritis (488-92, 822, 940-8, 1147). 1173



CHART XVII c
Trophic Disorders of Fat, Bone and Joints

Comprising Numbers 1124 to 1127 on left side of Chart
and 1175 to 1188 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

1124 FATTY TISSUE.	1136 Atrophy.	One of the earliest symptoms of diabetes mellitus is an in- excess of fat had been deposited. Patients lose weight and urine shows the constant presence of sugar. Atrophy of
	1137 Hypertrophy.	Large and tender deposits of fat, in lumps or in layers, with Arms and legs painful and tender, especially in the acute frequently in middle aged women (often alcoholic or syph- ilic).
1125 BONE TISSUE.	Failure in development.	Many cases occur, either congenitally or acquired in early so that the individuals remain throughout life of abnormal due to atrophy or loss of function of the thyroid, or pituitary mally formed (simple dwarfs or decidedly undersized men) described elsewhere, under infantilism and mongolism (1135) (chondro-dystrophia foetalis) there is a dystrophy of the normally in length; so that dwarfism results. The head is especially their proximal segment, the hand is short, the lordosis, pelvis contracted, legs often bowed or knock-kneed developed. Adults, as well as children, not infrequently in the legs, as in rickets, osteitis deformans (1182), osteo- kyphosis, etc.
	1138 Atrophy.	In cases of extensive acute anterior poliomyelitis and of cerebral or very slow growth of the part from disuse. One side of the face is much smaller than the other, due to fat. The process is usually progressive. It seems to be neuritis. Dryness, scaliness and loss of color of the skin small area atrophies, which atrophy gradually extends in process continues until the entire half of the face is atrophic parts of the body. One side of the tongue is usually atrophic the atrophy.
	1139 Hypertrophy.	One side of the face is much larger than the other, due to en- progressive, and seems in some cases to be due to a per- The bones of the head and face are enlarged, diffusely or not them. Headache, neuralgia, blindness, deafness and fingers are not enlarged. Forehead is bulging and head is often Disease commences late in life with slight pains, especially jaw is not enlarged. The head enlarges, the legs and vertebrae patients become shorter (even as much as a foot or more) Symmetrical enlargement of all the tissues, but especially the etc. It comes on gradually, patient requiring larger and larger shouldered" (kyphosis). These changes are often associated head and joints is a common symptom. The disease is in early life, before the epiphyses are joined by bone to bone The hands and feet are enlarged, and the fingers and toes "en- by the X-ray. These symptoms are associated with chro- toms vary greatly in degree and extent; the mildest form
	1140 Fragility.	In some persons the bones are unusually brittle and break of these cases occur in old age (senility), others occur in malacia), while others occur in children. The disease is thyrosis, etc.
1126 JOINT DISEASE.	Joints painless, enlarged, abnormally movable, especially hyperextension, cartilages eroded, effusion of synovial fluid, exostoses of bone. The exciting cause for these changes is often painless traumatism, at least in part.	Joint involvement not uncommon. Usually in legs. Joint involvement rare. Usually in arms.
1127 OTHER TROPHIC LESIONS.	1141 Atrophy and hypertrophy.	Atrophy or hypertrophy of different organs (mammary glands) requently met with and may be due to disordered nervous system.

OF SYMPTOMS

FAT AND BONE

SYMPTOMS

to deposit fat in the tissue, although previously often an long continued become emaciated. Examination of the occur in fevers and in many other conditions.

arms and legs. Face, feet and hands not much involved. as being deposited. Locomotion impeded. Occurs most

ony framework of the body does not develop normally; There is reason to believe that some of these cases are of these individuals are merely small but otherwise nor- now many physical deformities. Some cases have been 0, 1164) and microcephaly (1084). In *Achondroplasia* es, in consequence of which the bones do not increase e bridge of the nose depressed, the arms and legs short, ost equal length and divergent (trident shape), lumbar aormally lax. The muscles are rather unusually well nsequence of excessive bowing of weakened long bones and in consequence of curvature of the spine, as in

1 occurring in infancy there is often an arrest of growth

ssues, even of the bones, and especially of the skin and action, or cold and in some cases is due to a trigeminal oms. The process commences in the skin, of which a and inward to the fat, muscles and even bones. The ses, extends beyond the median line and even to other ie trigeminal nerve usually precedes and accompanies

tissues, especially of the bones. The process is usually

use pressure symptoms on the nerves running through us, common symptoms. Lower jaw and extremities

of the body become enlarged and soft, but the lower e bent and bowed (spondylitis and kyphosis). The fected.

and feet, lower jaw, and sternum, also ears, tongue, es. Thorax is much enlarged and patient is "round hemianopia, followed at times by blindness. Pain in ay of the pituitary body. If the disease commences d of acromegaly results.

of the forearms are also often enlarged, as can be shown of a septic or tuberculous nature usually. The symp- ers."

ence, even on turning the patient over in bed. Some ening of the bone and diminution of lime salts (osteo- variously named: osteogenesis imperfecta, osteopsa-

absent. Pains in legs. Ataxia without paralysis. oms. Argyll-Robertson's pupil reflex.

aggerated. Pains in arms. Paralysis of arms (slight). l and thermic, with persistence of tactile, sensibility.

ther parts of body (hands, fingers, etc.), are not infre- obscure significance and are without diagnostic value.

DIAGNOSIS

Diabetes Mellitus (900, 1172). 1175

Adiposis Dolorosa. Dercum's Disease (1012). 1176

Dwarfism, Microsmia, Nanosmia, Achondroplasia (1164). 1177

Disuse from Paralysis. 1178

Facial Hemiatrophy. 1179

Facial Hemihypertrophy. 1180

Hyperostosis Cranii or Leontiasis Ossea. 1181

Osteitis Deformans. Paget's Disease. 1182

Acromegaly and Gigantism. 1183

Hypertrophic Pulmonary Osteoarthropathy. 1184

Fragilitas Ossium. Osteopsathyrosis. 1185

Arthropathy of Tabes (661). (Charcot's Disease.) (Figs. 24-7.) 1186

Syringomyelia (552, 693, 1170). (Figs. 24-7.) 1187

Localized Hypertrophies and Atrophies, symmet- rical and asymmetrical. 1188



CHART XVII d
Ganglionic Disorders, Vaso-Motor Disorders

Comprising Numbers 1128, 1129 and 1142 to 1145 on left side of Chart
and 1191 to 1203 on right margin

DIAGNOSTIC

GANGLIONIC AND

ABSTR.

DIAGNOSTIC SYMPTOMS AND TESTS

1128 GANGLIONIC DISORDERS.

1142
Paralytic.

Ptosis of eyelid, although patient can raise it perfectly by an effort not dilate when shaded, although it contracts briskly when eye is sure with retraction and lowering of eyeball (enophthalmus). abolished, flushing of skin and absence of sweat on the affected side third rib.

1143
Irritative.

The symptoms are exactly opposite to those of paralysis of the eye widening of the palpebral fissure (Stellwag's sign) and delayed de The Boston-Kocher's sign, an amplification of Graefe's sign, may

Exophthalmus, tachycardia, goitre, flushing, sweating, tremor, nerve ward (Graefe's sign), widening of the palpebral fissure (Stellwag's The disease occurs much more frequently in women than in men the cervical sympathetic ganglia, yet it is really due to excessive the reverse of those of myxedema (1163), can be produced by th extirpation of the thyroid.

Paroxysmal spasm or congestion of the bronchioles, often reflex fr nervous temperament of most asthmatics, together with the very may be due to a disturbance of the thoracic sympathetic. The prolonged expiratory murmur, make the diagnosis easy. Asthma be in part voluntary, in part reflex; also is usually associated wit

Paroxysmal attacks of coldness and pallor ("dead fingers," "local s of all together. These attacks may last a few minutes or hours the same parts become dusky blue, or purplish black ("local asph This attack may pass off, after several hours, with abundant sw grenous and finally slough off. The necrosis does not usually in metrical. It is more common in cold weather and is often brou Hematuria and evidence of congestion of other internal organs n

Analogous to Raynaud's disease is gangrene of extremities occurin old age; either without the local syncope or local asphyxia, or w

Paroxysmal attacks of formication, tingling, numbness and other intervals and exclusively in women. They seem to be brought cases during the attack the skin becomes pale and blue. Similar

1144
Vascular.

Paroxysms of severe pain in one foot, rarely in both, rarely in han creased by allowing foot to hang down, or by motion of it, or by redness and swelling of the whole, or part, of the sole of foot. to a simple vaso-motor neurosis. The neuritis, when present, is

1129 VASO-MOTOR DISORDERS.

Occurs in middle aged or elderly persons and is associated with arter walk and increases so that walking becomes impossible. It passe attack the feet are cold and there is absent or greatly diminished alcohol and tobacco and injury seem to be common causes of this feet. The arms are rarely involved.

In many diseases if lines or writing be traced on the skin with a sha to lines of bright redness, which persist for minutes or hours.

1145
Exudative or
Secretory.

Paroxysmal attacks of localized edema of sub-cutaneous or sub-mu a few hours or days. The extent of the edema varies greatly. extremity, or even more. It may cause death when occurring in sure. They occur in hysteria and are usually associated with a are associated with symptoms of digestive disorder, they are calle except the itching. The disease often shows a strong heredity at

Edema of the legs, unilateral becoming bilateral, bad heredity. T being a sudden demarcation at the level of the joint. The edema

Some cases present paroxysmally or constantly a profuse sweating,

SYMPTOMS

R DISORDERS

TOMS

osis). Contraction of pupil (myosis), which does and on convergence. Narrowing of palpebral fissure diminished. The cilio-spinal reflex (335) is on side of neck, or of arm and thorax above the

. Dilatation of pupil (mydriasis), exophthalmus, lid when eye is turned downward (Graefe's sign). ease and in exophthalmic goitre (1193).

descent of upper eyelid when eye is turned down-systolic murmur in vessels of neck and in thyroid. ny of its symptoms may be referred to disorder of thyroid gland. Many of its symptoms, which are of thyroid gland, and the disease can be cured by

Freedom from symptoms in the interval. The cessation of the attack, indicates that the disease ks of dyspnoea, with the abundant dry rales and strong contraction of the diaphragm, which may

ling of fingers or toes or tip of nose or of ears or ss off, or may be followed by an attack, in which), from congestion. This is associated with pain. ts, or a small portion of them, may become gan- of the cyanotic area. The disease is usually sym- hands in cold water, or by working with hands. attacks.

ers of a family at varying ages from childhood to lications of these conditions in some of the cases.

ngers and hands. The attacks occur at irregular nd by having the hands in cold water. In some nes occur in the early stages of acromegaly (1183).

in face, lasting a few minutes or a few hours, in- except in the earliest attacks, is accompanied by en only, and is generally due to a neuritis, rarely with atheromatous arteries.

inful cramp occurs in muscles of legs after a short rest to return if walking is resumed. During the orsalis pedis or posterior tibial artery. Syphilis, disease not infrequently precedes gangrene of the

appear for a few seconds white, but soon change

ng localized swellings, either white or red, lasting lf inch in diameter, or may extend over an entire e swellings are not tender and do not pit on pres- sion. If the swellings are red in color, itch and sharp line can be drawn between the two diseases to be malarial.

limited above by the ankle, knee or groin; there d with pyrexia or gastric disturbance.

ometimes general.

DIAGNOSIS

Paralysis of Cervical Sympathetic.	1191
Irritation of Cervical Sympathetic.	1192
Exophthalmic Goitre.	1193
Asthma (617).	1194
Raynaud's Disease. Symmetrical Gangrene (1011).	1195
Family Gangrene.	1196
Acroparesthesia.	1197
Erythromelalgia (1010).	1198
Intermittent Limping or Claudication. Dysbasia Angio-Sclerotica (554).	1199
Dermographia (326, 1167).	1200
Angio-Neurotic Edema and Urticaria. (1167). Quincke's Disease.	1201
Milroy's or Meig's Disease. Trophedema.	1202
Hyperhidrosis. Excessive Sweating	1203



CHART XVIII
Syphilis of the Nervous System

Comprising Numbers 1205 to 1217

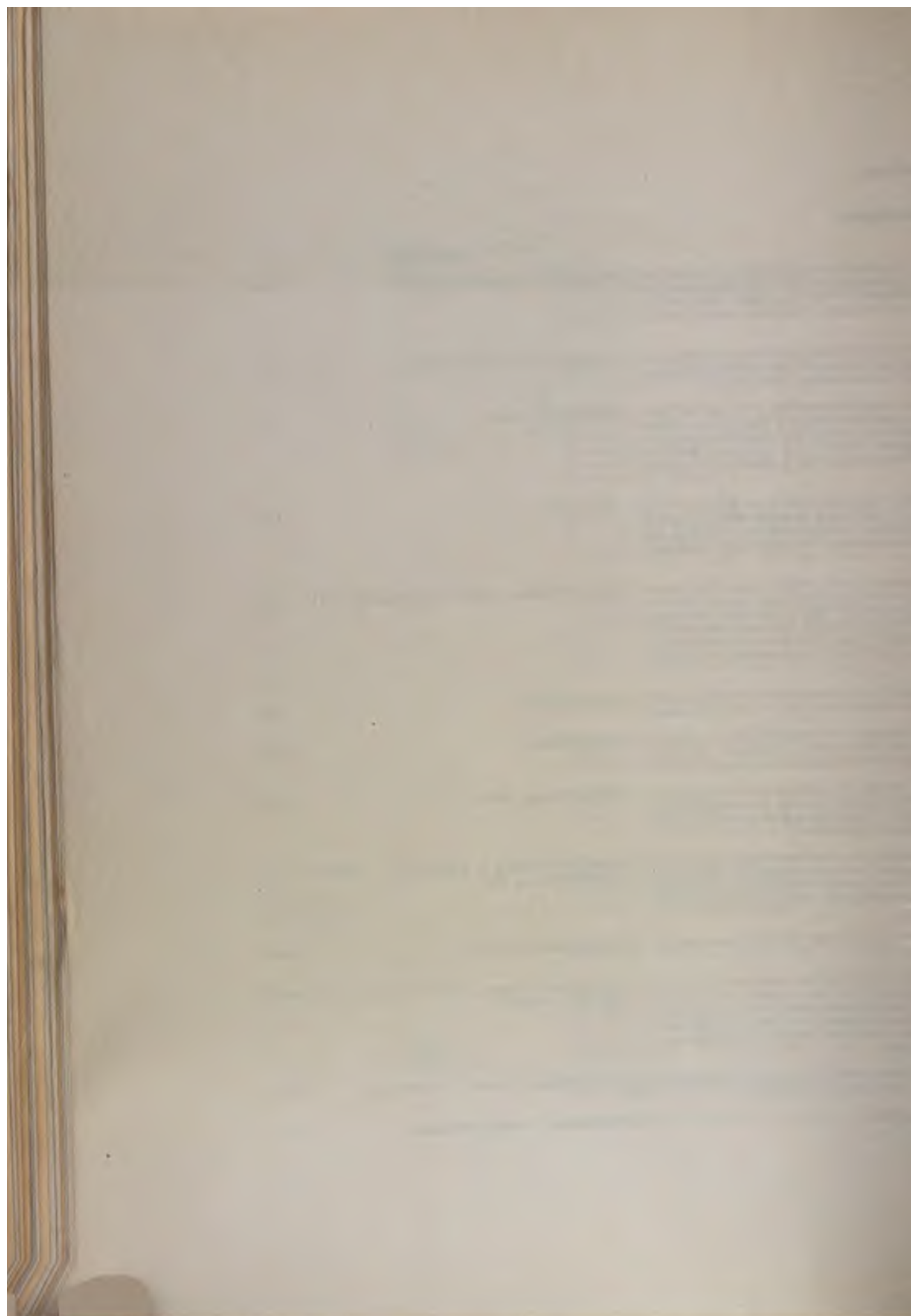


CHART XVIII
Syphilis of the Nervous System

Comprising Numbers 1205 to 1217

DIAGNOSTIC SYMPTOMS AND TESTS

1205

SYPHILIS OF THE
NERVOUS SYSTEM.

History of personal, or hereditary, syphilis. Physical evidence of syphilis; such as Wassermann reaction, a chancre or its scar, induration, mucuous patches, a syphilitic rash or its copper colored scars, hazy cornea, notched teeth, furrows about angle of mouth, saddle nose, ptosis, iritis, enlarged glands, periosteal nodes, etc.

Syphilitic Nervous Diseases.

Symptoms of syphilis of the nervous system are very variable from day to day, transitory and manifold.

They consist of paresis, rather than of complete paralysis. They usually show rapid improvement under K.I. and Hg. or Salvarsan. Nocturnal headache is common, as are also the Argyll-Robertson's pupillary reflex, unequal pupils and optic neuritis.

Cerebral symptoms.

Although these symptoms can be divided into several, more or less well defined, groups, yet a combination of several or all of the lesions, in varying intensity, is not infrequent; so that a combination of the symptoms of several or all of the groups may be present in one case. Pure, uncomplicated cases of each type are, however, commonly met with.

Spinal symptoms.

(Both forms of spinal syphilis may occur together.)

Cerebral and spinal symptoms.

Local peripheral symptoms.

Post-, or Meta-, syphilitic nervous disease.

Cerebral symptoms.

Spinal symptoms.

S OF SYMPTOMS

ROUS SYSTEM

ABSTRACT OF SYMPTOMS

DIAGNOSIS

ytosis from	Symptoms of cerebral tumor (507, 536). Other syphilitic symptoms may be present. Rapid course with irregular remissions and intermissions. The symptoms of cerebral compression are much less pronounced than in non-syphilitic tumors. Very amenable to anti-syphilitic treatment.	Isolated Cerebral Gumma.	1206
	Symptoms of cerebral thrombosis (506). The attacks occur rather early in adult life. There are many prodromata. Nocturnal headache is common. The paralysis is moderate in degree, variable in intensity and often temporary. Mental derangements, often in the form of trance-like states, frequently occurs. Branches of the basilar artery are involved most frequently, and the attack often occurs during sleep, or without coma during the day.	Cerebral Syphilitic Endarteritis and Thrombosis.	1207
mph- ebro- mbar	Symptoms of meningitis (590, 608), which may be very slight and very variable. With severe headache (nocturnal) there may be some nausea and vomiting. Little or no elevation of temperature or retraction of neck. No tuberculin reaction or evidence of tuberculosis. This disease is rare in children.	Syphilitic Meningitis of Convexity of Brain.	1208
	Symptoms of cortical irritation (Jacksonian epilepsy, local headache and tenderness) and paralysis of cortical functions (aphasia, monoplegia, etc.). Mental derangement is common, and often takes the form of paresis (pseudo-paresis), but is amenable to anti-syphilitic treatment.	Syphilitic Meningitis of Base of Brain, including Kahler's Disease.	1209
or no	Symptoms of Brown-Séquard's paralysis, or later of paraplegia (442, 509, 840, 981).	Isolated Spinal Gumma.	1210
	Symptoms of myelomalacia (485, 513-4, 517-8, 549-50).	Spinal Syphilitic Endarteritis and Thrombosis.	1211
	Symptoms of lateral sclerosis (525). (Fig. 26.)	Erb's Syphilitic Lateral Sclerosis.	1212
mph- ebro- mbar	Symptoms of spinal meningitis, or of pachymeningitis (550, 608, 974, 1005). Rigidity of back. Girdle pains and radiating pains, exaggerated reflexes in legs. Some of these cases present the symptoms of progressive spinal muscular atrophy (547).	Syphilitic Meningitis of Cord and of Nerve Roots. (Meningo-myelitis, Pachymeningitis Cervicalis Hypertrophica.)	1213
mph- al	A combination of the above symptoms (1208-9, 1213) in very varying extent and intensity. A clinical picture comprising cerebral and spinal symptoms and presenting great variations, which are impossible to describe in a few words.	Cerebro-Spinal Syphilis.	1214
the spinal	Symptoms of neuritis (488-92, 822-3, 940-8).	Syphilitic Neuritis.	1215
is in	Symptoms of general paresis (1104).	Paresis.	1216
	Symptoms of locomotor ataxia (661).	Locomotor Ataxia. Tabes. (Fig. 27.)	1217

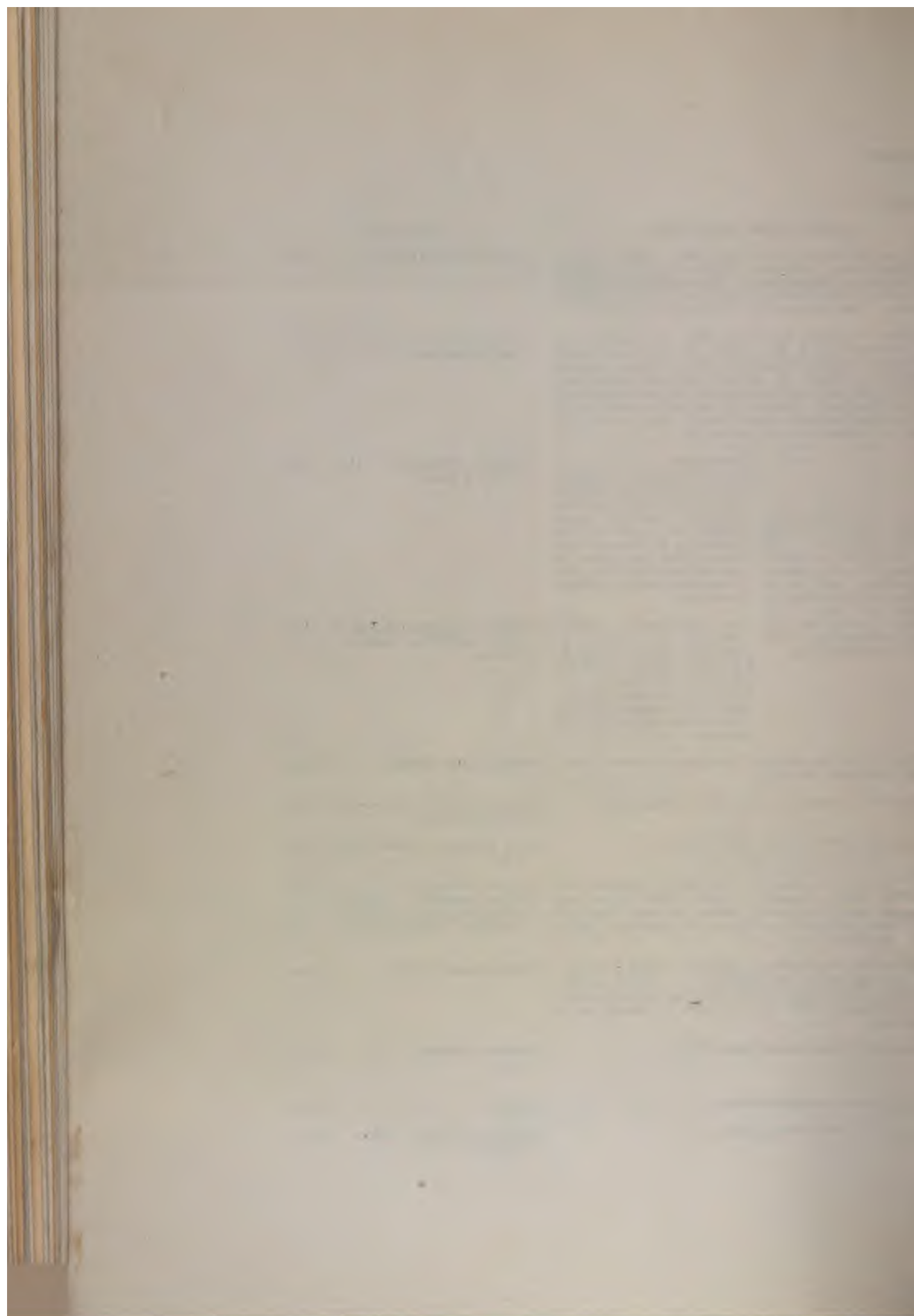


CHART XIX
Abnormal Cerebro-Spinal Fluid

Comprising Numbers 1220 to 1242

DIAGNOSTIC A

ABNORMAL C

TESTS AND

1220 ABNORMAL CEREBRO- SPINAL FLUID.

1221 Butyric acid test positive.	1223 Leucocytosis.	Weichselbaum's diplococcus intra-cellularis meningitidis or rarely Pneumococcus	Fluid may Tension is
		Weichselbaum's diplococcus. Pneumococcus, Pfeiffer's bacillus, Streptococcus, Staphylococcus, Typhoid bacillus, Bacterium coli, etc.	Fluid usu under high
	1224 Lymphocytosis.	Tubercle bacillus.	Fluid usu cate coag high tensi
		Tubercle bacillus.	
		Wassermann reaction posi- tive.	Fluid ele bacteria.
		Wassermann reaction nega- tive.	Tension is but not v
1222 Butyric acid test negative.	1225 No Lymphocytosis or Leucocytosis.	No bacteria and Wassermann negative.	Fluid ele tension; in bloody

OF SYMPTOMS

SPINAL FLUID

STIC SIGNS

DIAGNOSIS

cloudy.	Occurs in epidemics.	Symptoms of epidemic Cerebro-spinal meningitis (591).	Epidemic Cerebro-spinal Meningitis.	1226
y and	Occurs sporadically.	Symptoms of sporadic or purulent cerebro-spinal meningitis (592).	Sporadic Purulent Meningitis.	1227
th deli- under	{ Acute course. Chronic course.	{ Symptoms of tuberculous meningitis (593).	{ Acute, or sub-acute Tuberculous Meningitis.	1228
			{ Chronic Tuberculous Meningitis.	1229
	{ Tremor and mental symptoms.	Symptoms of Paresis (1104).	Paresis.	1230
from	{ Ataxia.	Symptoms of Tabes (661).	Tabes.	1231
	{ Symptoms not typically characteristic of paresis or tabes, being due to a cerebro-spinal meningitis.		Cerebro-spinal Syphilis (1208-9, 1213-14).	1232
	{ Motor paralysis.	Symptoms of acute anterior poliomyelitis (495). (Figs. 26-7.)	Acute Anterior Poliomyelitis.	1233
	{ Herpetetic rash.	Symptoms of herpes zoster (1166).	Herpes Zoster.	1234
reased	{ Epidemic. High fever.	Symptoms of Typhus.	Typhus Fever.	1235
	{ Choked disc usually present.	Symptoms of cerebral or spinal tumor (507, 578, 587).	Tumor.	1236
	{ Choked disc may be present.	{ Symptoms of cerebral or spinal abscess (508, 578, 587). Symptoms of hydrocephalus (411, 905, 960).	{ Abscess. Hydrocephalus.	{ 1237 1238
	{ Headache.	Symptoms of serous meningitis (594).	Serous Meningitis.	1239
	{ Apoplexy.	Symptoms of cerebral or spinal hemorrhage (503, 524, 1060-1).	Hemorrhage.	1240
reased often	{ Albumen and casts.	Examination of the urine shows albumen and casts. Edema, headache, dyspnoca, etc., usually present.	Uremia.	1241
	{ Anemia.	Examination shows anemia, pallor, etc., or acute infections, or some similar conditions.	Anemia.	1242

PART III

LOCALIZATION

OF

LESIONS WITHIN THE NERVOUS SYSTEM

BY

A CONSIDERATION OF THE
PARALYTIC AND IRRITATIVE SYMPTOMS
RESULTING FROM THEM

CHART XX
Spinal Localization
Comprising Numbers 1250 to 1267

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman

SEGMENT INVOLVED	MOTOR CONDITIONS			REFLEX CONDITIONS	SENSORY CONDITIONS	
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1256 IV Lum- bar	Muscles of rectum and anus. Muscles of bladder. Muscles of genitals. Obturator internus. Pyriformis. Gemelli. Gluteus medius. Gluteus minimus. Gluteus maximus. Biceps femoris. Semi-membranosus. Semi-tendinosus. Popliteus. Gastrocnemius. Soleus. Flexors of toes. Extensors of toes. Peroneus brevis. Peroneus longus. Tibialis anticus.	Obturator internus.	Defecation, with fecal incontinence. Micturition, with dribbling. Erection and ejaculation impossible. Outward rotation of thigh weak. Inward rotation impossible. Retraction of thigh impossible. Flexion of knee lost. Plantar flexion of foot lost. Flexion and extension of toes lost. Raising outer margin of foot. Raising inner margin. Extension of thigh weak. Adduction difficult.	Patellar may be wanting.	Plantar.	As above, and inner side of lower legs and dorsum of feet, and strip on outer posterior surface of thighs.
1257 III Lum- bar	Muscles of anus, bladder and genitals. Outward rotators and thigh. Inward rotators of thigh. Retractor (flexor) thigh. Flexors of knee. Plantar flexors of foot. Flexors of toes. Extensors of foot. Vastus externus.	Vastus internus. Rectus femoris. Crureus. Adductors of thigh. Flexors of thigh at the hips.	All movements of legs are lost, except that extension of legs is barely possible and that the thigh can be flexed on body by the psoas and iliacus. Defecation and micturition are destroyed. Urine and feces dribble and cannot be retained.	Patellar and cremasteric.	Ankle-clonus may exist.	As above, and whole of legs except a triangular area on front of thigh with base at Poupart's ligament.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman						
SEGMENT INVOLVED	MOTOR CONDITIONS			REFLEX CONDITIONS	SENSORY CONDITIONS	
	Paralysis	Paresis	Actions lost or impaired	Absent	In-creased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1258 II Lum- bar	Paralysis of all muscles of lower extremity, except psoas.	Psoas.	Complete paralysis of legs, rectum and bladder. As above.	Patellar, Achilles, and cremas-teric.	Achil-les may be in-creased. Plantar.	Whole of legs and pelvis. (Testicles not sensitive to pressure.)
1259 I Lum- bar	Total paralysis of whole lower extremity, psoas included.			Crema-steric and Achilles.	Patel-lar ab-sent or in-creased.	As above, and groins and front of scrotum and penis.
1260 XII to III Dor- sal	Paralysis of lower extrem-ity, and gluteal region. Paralysis of abdominal and dorsal regions, gradu-ally added as the site of the lesion ascends.		As above, and paralysis of mus-cles of respiration causes diaphragm-atic breathing and dyspnoea.	Epigas-tric and umbilical reflex.	Patel-lar, cre-mas-teric, Achil-les and Plan-tar.	As above, and a band running around body about two seg-ments below the one in-volved and limited above by a narrow zone of hyper-esthesia.
1261 II Dor- sal	As in 3d dorsal.		As above.	All below lost in complete division of cord.	All subja-cent re-flexes.	As above, and a strip on the inner side of the upper arms.
1262 I Dor- sal	All muscles of trunk and lower ex-tremities.	Flexion of fingers. Muscles of the little finger. III and IV inter-ossei. Lumbricales. Pronator quadratus. Lower part of pec-toralis major. Lower part of pec-toralis minor.	As above and weak-ness in flexion of fingers. Pronation dis-turbed.	Oculo-pupillary symp-toms. All below lost in complete division of cord.	All subja-cent re-flexes.	As above, and a strip on the inner side of the forearms.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman						
SEGMENT INVOLVED	MOTOR CONDITIONS			REFLEX CONDITIONS	SENSORY CONDITIONS	
	Paralysis	Paresis	Actions lost or impaired	Absent	In-creased in partial lesions	
1263 VIII Cervical	Paralysis of muscles of trunk and lower extremities.	Flexors of the little finger.	As above.	Oculo-pupillary symptoms.	All below.	As above, and the fingers, except volar surface of the thumb and the ulnar surface of the little finger.
	Abductor of little finger.	Opponens minimi digiti.				
	Adductor of thumb.	Flexor subl. digitorum.				
	Flexor of the little finger.	Flexor profun. digitorum.		All below lost in complete division of cord.		
	Opponens minimi digiti.	Flexor carpi ulnaris.	Hand weak.			The cervical sensory nerve roots supply the same area of the skin in common, especially in the hands and fingers. Hence the anesthesia is slight and uncertain.
	III and IV interossei.	Extensors of the thumb and fingers.	Extension of arm.			
	Lumbricales.	Triceps (slight).	Int. rotation and retraction of arm.			
		Latissimus dorsi (lower part).	Adduction of arm.			
		Pectoralis major.				
		Pectoralis minor.				
1264 VII Cervical	Lower extremities and trunk.	Extensors, Flexors and Abductors of thumb.	As above and Hand very weak. (Winged scapulae.)	Arm reflexes.	All below.	As above, and most of the hands and a small strip on the anterior, another on the posterior, surface of the forearm.
	Flexor profundus digitorum (ulnar side).	Extensor indicis.	Retraction and inward rotation of arm.	Forearm reflexes.		
	Flexor carpi ulnaris.	Extensors of the fingers (movements barely possible).		Palmar reflex.		
	Small hand muscles.	Supinator longus.				
	Pronator quadratus.	Biceps (very slightly parietic).		All below lost in complete cord division.		
		Triceps.				
		Pectoralis major.				
		Serratus magnus (slight).				
		Latissimus dorsi.				
		Teres major.				

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF THE CORD (Continued)

Modified from Wichman

SEGMENT INVOLVED	MOTOR CONDITIONS			REFLEX CONDITIONS		SENSORY CONDITIONS Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
	Paralysis	Paresis	Actions lost or impaired	Absent	In-creased in partial lesions	
1265 VI Cervical	Muscles of lower extremity and trunk. Muscles of fingers (including thumb) and hand. Triceps. Pectoralis major. Latissimus dorsi. Teres major. Infraspinatus. Serratus magnus.	Coraco-brachialis. Biceps. Brachialis anticus. Supinator brevis. Deltoid. Scaleni. Splenii. Deep head and neck muscles.	As above and movements of fingers and thumb impossible. Extension of forearm. Flexion of forearm weak. Supination very weak. Adduction of arm and inward rotation. Adduction, retraction and external rotation. "Winged" scapulae. Raising of arm. Rotation of head. Fatal in a few days or weeks.	Arm reflexes. Extensor forearm reflexes. All below lost in complete cord division.	All below.	As above, and whole of hands and fingers and radial side of forearm.
1266 V Cervical	Muscles of lower extremities and trunk. All the muscles of the arm, forearm, hand and fingers; even the deltoid, coraco-brachialis and brachialis anticus. Deep cervical muscles. Intercostals.	Levator anguli scapulae. Scaleni. Diaphragm (because of filaments from V cervical segment to phrenic nerve), or spread of injury from 5th to 4th cervical segment. Trapezius and sterno-cleido-mas-toid are intact.	As above and shoulders raised with difficulty. Rotation and flexion of head. Dyspnoea. Fatal in a few hours or days.	Scapular and tendon reflexes of paralysed muscles in arms. All below lost in complete cord division.	All below.	As above, and whole of arms, except tip of shoulder.
1267 IV-I Cervical	Total cross-lesions from the fourth cervical segment upward are rapidly fatal, because of complete paralysis of the diaphragm and intercostals.					
	Total cross-lesions of the brain-stem are rapidly fatal for the same reason.					

CHART XXI
Cerebral Localization
Comprising Numbers 1268 to 1286

CHART XXI a
Cerebral Localization in the Medulla and Pons:
Ganglia at Base

Comprising Numbers 1268 and 1269

TABLE OF SYMPTOMS IN THE

SEAT OF LESION		PARALYSIS OF MOTION	PARALYSIS OF SENSATION	LOCALIZATION	ACTION LOSS OR IMPAIRMENT
1268	Lesion involving lateral half of the Medulla Oblongata.	Crossed paralysis: hemiplegia alternans hypoglossica. Homolateral half of tongue, diaphragm and vocal cord, contralateral arm and leg. In some cases arm and leg may be paralysed on both sides, but not equally so. Extremely rarely leg on one side and arm on the other are paralysed.	Taste in posterior part of homolateral half of tongue. All forms of sensation in pharynx and throughout the respiratory tract. Analgesia and thermic anesthesia of homolateral half of face and contralateral half of body. Anesthesia of one side, or of both sides of the body.		Articulation, deglutition, peristalsis, cardiac action, vomiting, use of arms and of one or both sides
Babinski and Nageotte's Bulbar Syndrome (437). Rare because of the small transverse area of the medulla. Thrombosis of Posterior Inferior Cerebellar Artery causes very similar symptoms. (Figs. 21-3.)					
1269	Lesion in Lower (Caudal) Third.	Confined to the bridge portion.	Crossed paralysis: hemiplegia alternans facialis. Muscles of expression of homolateral half of face and the external rectus at times, and contralateral arm, leg and half of tongue (Millard-Gubber's syndrome—439).	None, unless indirectly from pressure and then contralateral hemianesthesia.	Articulation, mastication. Movement of homolateral face, and of contralateral arm and leg.
		Confined to the tegmentum.	Muscles of expression of homolateral half of face and of external rectus (Foville's paralysis). Contralateral arm and leg may be slightly involved.	Contralateral hemianalgesia and thermic anesthesia and at times hemianesthesia. Anesthesia, and especially analgesia, of homolateral half of face (Hemianesthesia alternans). Very rarely, deafness. Rarely dissociation of sensation.	Articulation, mastication. Movement of homolateral half
	Lesion in Middle and Upper (Cephalad) Third.	Confined to the bridge portion.	Complete contralateral hemiplegia.	Usually of all forms of sensation in homolateral half of face. Occasionally also hemianesthesia of contralateral half of body.	Chewing and articulation. Movement of contralateral body.
		Confined to the tegmentum.	Conjugate deviation of eyeballs toward the side of the lesion. May be complete hemiplegia of slight degree from pressure.	Paralysis of all forms of sensation on homolateral half of face. Contralateral hemianalgesia. May be contralateral hemianesthesia.	Chewing and articulation. Movement of toward the same side as the lesion.

SE LESIONS OF BRAIN-STEM

ULLA AND PONS

REFLEXES ALTERED	VERTIGO	ATAXIA	MUSCLE SENSE	SECRETORY AND OTHER DISTURBANCES
Tendon reflexes increased with Babinski and ankle-clonus on opposite side. Cutaneous reflexes may or may not be increased.	Usually present.	Usually present and of both motor and cerebellar type. Homolateral.	Usually lost, especially if motor ataxia be present.	Myosis and pseudo-ptosis (ophthalmoplegia sympathica) and salivation are common. Cheyne-Stokes's respiration (435).
Tendon reflexes increased with Babinski and ankle-clonus on opposite side. Cutaneous reflexes may or may not be increased.	Often present.	No motor, but there may be cerebellar, ataxia.	Normal.	Conjunctivitis is frequent in eye of same side. May be a tendency to fall or to turn to one side. Salivation.
Normal or slightly exaggerated as above.	Usually present.	Usually present on the same side as the lesion.	Lost on the same side as the lesion.	Conjunctivitis is frequent in the eye of the same side. Salivation.
Tendon reflexes increased with Babinski and ankle-clonus on the opposite side. Cutaneous reflexes may or may not be increased.	Often present.	No motor, may be cerebellar, ataxia	Normal.	Ulceration of cornea may occur. May be a tendency to fall or turn to one side.
Normal or may be slightly exaggerated.	Present.	May be motor and cerebellar ataxia.	Lost on the same side as the lesion.	Ulceration of the cornea may rarely occur. A slow rhythmic tremor of the arm and leg of opposite side may be present.

CHART XXI b

Cerebral Localization: Ganglia at Base

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF BRAIN STEM AND CEREBELLUM

SEAT OF LESION	PARALYSIS OF MOTION	PARALYSIS OF SENSATION	ACTIONS LOST OR IMPAIRED	REFLEXES ALTERED	VERTIGO	ATAXIA	MUSCLE SENSE	SECRETORY AND OTHER DISTURBANCES	
1270 Crura Cerebri	Lesion confined to the pes or foot.	Some, or all, of the ocular muscles (except external rectus) on the same side, combined with a contralateral hemiplegia, usually complete. Hemiplegia alternans oculomotoria. (Weber's syndrome, 440).	None.	Movement of eyeball. Use of contralateral half of the body.	Tendon reflexes increased, with Babinski and ankle-clonus, on opposite side. Cutaneous reflexes may or may not be increased.	Usually absent.	None.	Normal.	Tremor resembling that of paralysis agitans (Benedikt's syndrome). (441)
	Lesion confined to the tegmentum.	One or more ocular muscles, except the abducens.	Contralateral hemianesthesia, or hemianalgesia and thermic hemianesthesia, or both. Deafness may be present, if lesion be bilateral.	Movement of eyeball.	Tendon reflexes normal.	Present.	Cerebellar type.	Impaired.	A slow, rhythmic tremor of arm and leg of opposite side may be present.
1271 Corpora Quadrigemina.	Lesion confined to anterior pair (nates).	Bilateral, more or less extensive, of all ocular muscles, except the abducens.	May be blindness without choked disc or other lesion.	Movement of eyeball.	Pupil reflex lost to both light and accommodation.	Usually absent.	May be absent.	Normal.	Nystagmus (at times vertical), squint, pupils often unequal.
	Lesion confined to posterior pair (testes).	None or may be slight paralysis as above, or of trochlearis.	May be deafness, if lesion be bilateral.	None, except chewing at times.	Normal.	Usually present.	Present. Of cerebellar type.	Normal.	May be slow, rhythmic tremor of arm and leg of opposite side, especially on voluntary motion.
1272 Cerebellum.	None.	None.	Walking and standing.	Normal or slightly exaggerated. Rarely abolished	Usually present.	Cerebellar ataxia with hypotonia almost always present.	Normal.	Nystagmus (80), tendency to fall to one side, occipital headache is frequent, cerebellar fits may occur.	
1273 Middle cerebellar peduncles.	None.	None.	Walking, standing and sitting.	Normal or slightly exaggerated.	Usually present.	Usually present with hypotonia of the cerebellar type.	Normal.	Tendency to fall or to turn eyes, head or body to one side. Rotatory movements, more or less pronounced, choreic spasms in homolateral half of body, and vertical divergence of the eyeballs sometimes occur.	
Lesions of inferior cerebellar peduncles cause lateropulsion; those of the superior cerebellar peduncles cause choreiform movements and cerebellar ataxia.									
1274 Base of Cranium.	Fractures, tumors, etc., at base of skull may cause many of the above symptoms according to their position, but their early and characteristic symptom is paralysis of one or more of the cranial nerves. Symptoms of paralysis predominate over those of irritation.								

Lesions of inferior cerebellar peduncles cause lateropulsion; those of the superior cerebellar peduncles cause choreiform movements and cerebellar ataxia.

Small lesions, not so extensive as to involve the entire lateral half of the brain stem, may occur at any point. The symptoms of these lesions depend upon the function (physiology) of the part affected and will naturally vary greatly. The location of such a lesion in a transverse section will depend upon what longitudinal fiber tracts are involved, and in longitudinal section upon what cranial nuclei and nerves parts are involved, as shown by the symptoms present in any case. A study of the figures at the end of this book is essential for the localization of such lesions and will serve this purpose better than a long verbal description.

CHART XXI^c

Cerebral Localization: Ganglia at Base

LOCALIZING SYMPTOMS IN LESIONS OF GANGLIA AT BASE OF BRAIN

SEAT OF LESION	DIAGNOSTIC SYMPTOMS
1275 Optic Thalamus. (Fig. 17)	Symptoms are variable and uncertain. May be hemianopia (pulvinar, and external geniculate involvement) with hemiopic pupillary reaction, contralateral hemianalgesia. Rigidity, choreiform movements, athetosis, and incoordination of contralateral leg, arm, and half of face may be present. The above mentioned motor disturbances occur also in lesions just external to the optic thalamus which involve the fibers connecting the thalamus with the cerebral cortex. Sensory disturbances (pain, hemianesthesia dolorosa, anesthesia, loss of muscle sense) may be present in the same parts. Occasionally a slight irritation of the skin is not felt at all, while a stronger one is felt inordinately. Absence of emotional expression in face, even when not paralyzed. Vaso-motor disturbances may occur in opposite side of body. Isolated analgesia or thermic anesthesia does not occur in lesions above the optic thalamus, but other forms of anesthesia do.
1276 Corpus Striatum. (Fig. 17)	<p>Nucleus Lenticularis and Nucleus Caudatus. No diagnostic symptom except the hemiplegia due to the involvement of the internal capsule. In rare cases a lesion of the nucleus lenticularis may be of such a form as to injure the anterior and posterior part of the posterior limb of the internal capsule, while its middle part escapes. In such cases there results a hemiplegia which involves the leg and face more than the arm. Dysarthria is a not uncommon symptom and in some cases the symptoms of sensory irritation: muscle spasm and incoordination described under lesions of the optic thalamus have been present. When the ganglia on both sides are affected, voluntary voiding of urine may be impossible, while automatic involuntary voiding may occur at regular intervals.</p> <p>Internal Capsule. Lesions in the anterior limb of the internal capsule cause either no symptoms or a paralysis of contralateral half of face. There may be ataxia and athetoid movements.</p> <p>Lesions in the anterior two-thirds of the posterior limb of the internal capsule cause a total contralateral hemiplegia of the body. This hemiplegia consists purely of a muscular paralysis and never produces a paralysis of the cortical functions such as aphasia, alexia, etc; but may produce dysarthria.</p> <p>Lesions in the posterior third of the posterior limb of the internal capsule cause hemianesthesia and loss of muscle sense on the opposite side of the body.</p> <p>Lesions at the extreme posterior end of the posterior limb of the internal capsule, in addition to hemianesthesia, cause contralateral hemianopia, deafness only if the lesion be bilateral and often the symptoms of motor irritation, described under lesions of optic thalamus.</p>
1277 Corpus Callosum.	No diagnostic symptoms.
1278 Island of Reil, Claustrum and External capsule. (Fig. 17)	Lesions in this area produce disturbances of speech, grouped under the general term paraphasia, and may produce anarthria, the result of complete aphasia.
1279 Pituitary Gland.	Hypertrophy, tumor, hemorrhage and some other lesions of the gland associated with excess of secretion may cause acromegaly or gigantism, in addition to a progressive bi-temporal hemianopia, terminating in blindness. A defect or atrophy of the gland associated with a diminution of secretion in early life may cause dwarfism and may produce pituitary eunuchismus or adiposogenital degeneration with excess of fat and a defect in the formation of the genitals. In any case of pituitary disease there may be polyuria, polydipsia and occasionally glycosuria and very rarely an escape of cerebrospinal fluid from the nose (hydrorrhoea nasalis). In some cases of pituitary disease there are no symptoms.
1280 Pineal Gland.	Abnormal growth of hair and deposition of fat. Abnormalities of genitals (at times with attacks of sexual excitement). Excessive growth in height of body (dyspinelismus). In consequence of involvement of adjacent tissue, bilateral ocular paralysis, nystagmus, pupil abnormalities, ataxia, and perhaps disturbances of hearing may be present.

CHART XXI d

Cerebral Localization: Lobes of Brain

LOCALIZING SYMPTOMS IN LESIONS OF CEREBRAL HEMISPHERES

SEAT OF LESION

DIAGNOSTIC SYMPTOMS

1282
FRONTAL LOBE
Contains the centers for all the skilled acts, especially the left lobe. Large lesions in the frontal lobes may cause a change in character and disposition of the patient. Many lesions, especially tumors, cause Jacksonian epilepsy, especially when situated in posterior part of lobe; while lesions in anterior part of lobe may cause epileptiform convulsions. Ataxia sometimes occurs in tumors in the frontal lobe. (Fig. 15)

The ascending frontal convolution.

Lesions in this region may cause awkwardness (cortical ataxia, apraxia) rather than paralysis.

The base of the middle left frontal convolution.

The base of the inferior left frontal convolution.

Lesion in the upper fourth of this convolution may cause Jacksonian epilepsy commencing in, and motor paralysis of, the contralateral leg. Very large lesions (hemorrhage, tumors, etc.) in this region may cause also paralysis of the homolateral leg in a lesser degree.

Lesions in the middle half of the convolution may cause Jacksonian epilepsy commencing in, and awkwardness of or loss of skill or complete paralysis of, the contralateral arm. Very minute lesions in the upper part of this region may affect only the shoulder; in the lower part, only the hand.

Lesions in the lower fourth of this convolution may cause Jacksonian epilepsy commencing in, and paralysis of, the contralateral half of face and neck. Very minute lesions in the upper part of this region, may affect only the eyes; in the lower and anterior part, the tongue and larynx.

Small lesions in this area may cause in right-handed persons, argaphia, and in many cases Jacksonian epilepsy, commencing in the contralateral arm.

Small lesions in this area may cause, in right-handed persons, motor aphasia, and in many cases Jacksonian epilepsy, commencing in the right side of the face.

1283
PARIETAL LOBE
Contains the centers for cutaneous and muscular sensation. Many lesions, especially tumor, cause Jacksonian epilepsy when situated in the anterior portion of this lobe; while lesions in posterior portion may cause epileptiform convulsions. (Fig. 15)

The ascending parietal convolution.

The left angular gyrus.

The rest of the parietal cortex.

Lesions in the upper fourth of this convolution may cause some blunting of cutaneous sensibility, and especially astereognosis in contralateral leg and foot.

Lesions in the middle half of this convolution may cause some blunting of cutaneous sensibility, and especially astereognosis in contralateral arm and hand.

Lesions in the lower fourth of this convolution may cause some blunting of cutaneous sensibility, and especially astereognosis in contralateral half of face.

Deep lesions in this region, in right-handed persons may cause alexia and hemianopia.

Lesions in this region may cause loss of muscular sense and motor ataxia in the contralateral arm and leg.

1284
TEMPORAL LOBE
Contains, on the left side, the centers of sensory speech. Lesions may cause epileptiform convulsions. (Fig. 15)

Lesions in the posterior portion of the left superior temporal convolution in right-handed persons, may cause sensory aphasia (psychic deafness).

1285
OCCIPITAL LOBE
Contains the centers of sight. Lesions may cause epileptiform convulsions. (Fig. 15)

Neighborhood of calcarine fissure.

Rest of occipital lobe.

Lesions in this area cause contralateral homonymous hemianopia. A lesion limited to the superior lip of this fissure causes quadrantic hemianopia or tetartanopia of the contralateral lower quadrants of field of vision. A lesion limited to the inferior lip of this fissure causes loss of contralateral upper quadrants of the field of vision.

Lesions in this area may cause loss of power of recognition of persons and things (psychic blindness).

1286
Cortical Lesions.
(Fig. 15)

Many lesions cause a mixture of paralysis and convulsions over a limited area which in some cases may slowly grow larger. The intelligence of the patient is always more or less impaired.

CHART XXII

Cerebro-Spinal Localization

TOPICAL DIAGNOSIS

LOCALIZATION OF LESIONS FROM ANALYSIS OF SYMPTOMS

1290 PARALYSIS The most important of all localizing symptoms.	1292 The reflexes in the paralysed area are abolished (except in 1310 and 1329) A lesion of the peripheral neurons.	1294 Sensation alone, in all its forms is lost or impaired.	} See Chart XXII a.
		1295 Motion alone is lost or impaired.	
		1296 Both motion and sensation are lost or impaired.	
	1293 The reflexes are present (except in 1357 and 1359) A lesion of the central neurons.	1297 Special forms of peripheral paralyses.	See Chart XXII b.
		1298 Sensory paralysis dominant. Little or no motor paralysis.	See Chart XXII c.
		1299 Motor paralysis dominant. Little or no sensory paralysis.	See Chart XXII d.
1291 Jacksonian Epilepsy, together with other symptoms of cerebral disease.	1300 Both motor and sensory paralysis well marked.		} See Chart XXII e.

For diseases and lesions accompanied by *motor paralysis* see 469, by *motor spasm* see 570, by *ataxia* see 638, by *tremor* see 639, by *nystagmus* see 640, by *fibrillation* see 641, by *local paralysis* see 636, by *local spasm* see 637, by *disorders of speech* see 735, by *disorders of gait* see 736, by *anesthesia and analgesia* see 810-12, by *disorders of special senses* see 807-9, by *pain* see 931, by *vertigo* see 932, by *mental disorders* see 1036, by *trophic disorders* see 1120, by *vaso-motor disorders* see 1129, by *ganglionic disorders* see 1128, by *syphilis* see 1205, by *abnormal cerebro-spinal fluid* see 1220.

CHART XXII a

Cerebro-Spinal Localization Paralysis with Abolished Reflexes

TOPICAL DIAGNOSIS

LOCALIZATION OF LESION FROM ANALYSIS OF SYMPTOMS

DIAGNOSTIC SYMPTOMS AND TESTS				LOCALIZATION			
REFLEXES ABOLISHED	1294 Sensation alone, in all its forms, is lost or impaired.	Area of anesthesia, etc. lies within the area of distribution of one or more nerves.	Onset acute or sub-acute.	Nerve involved, if palpable, is tender on pressure. No symptom of disease of central organs usually, unless nuclei are affected.	Lesion is in one or more sensory cranial nerves or nuclei or sensory end-organ; the nerve affected depending upon its anatomical distribution (822). (Figs. 19-21, 33, 38).	1301	
		Area of anesthesia, etc. lies within the area of distribution of one or more nerve roots.	Onset acute or chronic.	Nerves involved, if palpable, are not tender. May be symptoms of disease of central organs.	Lesion is in corresponding sensory nucleus in the brain stem, or in the posterior horn of spinal cord, or in column of Burdach, or in posterior nerve root. (Figs. 19-21, 24-6).	1302	
	1295 Motion alone is lost or impaired.	The paralysis is limited to muscles supplied by one or more nerves. (Figs. 19-21.)	Onset acute or sub-acute. No fever at onset.	Nerve involved, if palpable, is tender on pressure. No symptoms of disease of central organs. All the muscles supplied by the nerve are paralyzed, usually.	Lesion is in one or more motor cranial nerves, or a mild lesion of mixed spinal nerves; the nerve affected is the nerve supplying the paralysed muscles (489-93). (Figs. 19-21, 33, 38).	1303	
		The paralysis is limited to muscles supplied by one or more nerve roots. (Figs. 19-21)	Onset acute or chronic. May be fever at onset.	Nerve involved, if palpable, not tender. May be symptoms of disease of central organs. Often only a portion of the muscles innervated by the nucleus are paralyzed.	Lesion is in corresponding motor nucleus within brain stem, or in anterior horn of spinal cord, or in the anterior nerve root (493-5). Figs. 19-21, 24-6).	1304	
	1296 Both motion and sensation are lost or impaired.	U N I L A T E R A L	Motor and sensory paralysis is within the area of distribution of one spinal nerve.	Onset acute or sub-acute. No fever at onset.	Lesion in one spinal nerve (489). (Figs. 33, 38).	1305	
			Motor or sensory paralysis is within the area of distribution of several nerves from one plexus.	Nerve involved; tender on pressure. No symptoms of disease of central organs.	Lesion in brachial or lumbar plexus (490). (Fig. 32, 38).	1306	
		B I L A T E R A L	Motor and sensory paralysis extends over legs or arms or both, or even more generally.	Nerves involved tender on pressure. No symptoms of disease of central organs.	Muscles show weakness, tenderness and rapid atrophy.	Lesion of many spinal and (rarely) cranial nerves also (multiple neuritis) (488).	1307
			Onset acute or sub-acute. May be fever at onset.	Nerves involved not tender. There are disturbances of organic reflexes and other symptoms of organic disease of central organs.	Great pain. May be deformity of lumbar spines. Symptoms less symmetrical and bed-sores less common than in lumbar lesions. Domain of anterior crural nerve may be normal when lesion is low.	Lesion of 1308 cauda equina (487). (Fig. 29).	
				Legs alone are paralyzed and exhibit trophic disturbances. Anesthesia of rectum and bladder.	Little pain. May be deformity of lower dorsal spines. Symptoms symmetrical. Bed-sores always present. No portion of legs escape.	Lesion of lum- 1309 bar enlargement of spinal cord (484-7). (Fig. 24-6).	
				Both legs and arms are paralyzed. There are trophic disturbances in arms but not in legs. Reflexes are abolished in arms, exaggerated in legs (548-51).		Lesion of cer- 1310 vical enlargement of spinal cord (Fig. 24-6).	

CHART XXII b

Cerebro-Spinal Localization

**Comprising Numbers 1297 and 1315 to 1317 on left side of chart
and 1318 to 1336 on right margin**

TO
LOCALIZATION OF LES
PERIPHERAL PARAL
DIAGNOST

1297
SPECIAL FORMS OF
PERIPHERAL PAR-
ALYSIS. REFLEXES
ABOLISHED IN PAR-
ALYSED AREA, EX-
CEPT IN 1329.

1315
DISTURBANCES OF
VISION. (807.)

Blindness of entire field of vision of one eye is present.
Bitemporal hemianopia is present. The outer half is present.
Nasal hemianopia is present. The inner half is present.
Homonymous hemianopia is present. Identical pupillary reflex is present i.e. reflex is absent w

1316
PARALYSIS OF OCULAR MUSCLES (700).

All muscles of one eye paralysed. Eyeball protruded.
All muscles supplied by third cranial nerve are paralysed at once.
Partial or progressive paralysis of muscles supplied

No hemiplegia. Oculomotor.
Paralysis of arm and hand.
Tremor of arm and hand causing ataxia.

Paralysis of external rectus muscle.

No hemiplegia. Oculomotor.
Hemiplegia often associated with conjugate deviation.

Lower branch of facial nerve only, or mainly, paralysed.

Other symptoms of facial nerve lesion never present

1317
FACIAL PARALYSIS (703).

Both lower and upper branches of facial nerve equally paralysed.

Paralysis of arm and hand.
No hemiplegia. Chorea and abducens, maxillary.
Associated with unilateral deafness.
No deafness but hyperacusis, and often times absence of taste.
Hyperacusis. Loss of taste.
No hyperacusis. Loss of taste.
No hyperacusis. No loss of taste.

OSIS

ANALYSIS OF SYMPTOMS

ABOLISHED REFLEXES

S AND TESTS

LOCALIZATION

nerve is atrophied. Pupil does not respond to	Lesion in optic nerve (897-8).	1318
of vision is blind. Hemipic pupillary reflex	Lesion is in the central part of optic chiasm (362, 815, 860, 892).	1319
of one eye is blind. Hemipic pupillary reflex	Lesion is in outer margin of optic chiasm (362, 815, 861).	1320
(left) of each field of vision is blind. Hemipic half of retina is excited by light.	Lesion is in the optic tract or external geniculate body of opposite side (858, 893).	1321
evidence of disease within orbit.	Lesion within the orbit (914).	1322
ves paralysed.	Lesion of 3rd cranial nerve trunk or nucleus (700). (Fig. 18.)	1323
side.	Lesion involving crus cerebri (676).	1324
side present at rest and exaggerated on motion.	Lesion of red nucleus or rubro-spinal tract on same side as motor oculi paralysis (441, 676).	1325
ial nerve (700).	Lesion of 3rd cranial nucleus, in whole or in part (700). (Fig. 18.)	1326
ves paralysed, especially the facial.	Lesion of 6th cranial nerve or nucleus (1330-1). (Figs. 19, 20.)	1327
emianesthesia of opposite side. Loss of power o right or left. Facial or auditory nerve may	Diffuse lesion of Pons Varolii (539, 883). (Figs. 19, 20.)	1328
brain present. Electrical reaction of degenera- sent.	Lesion above nucleus of facial nerve in cerebral hemispheres or in crura cerebri. (Fig. 15, 19).	1329
side. Often abducens paralysis.	Lesion in Pons Varolii. (Figs. 19, 20.)	1330
ually. Other cranial nerves, especially auditory	Lesion of nucleus of facial nerve. (Figs. 19, 20).	1331
and vertigo without disease of the ear.	Lesion of facial nerve trunk at base of brain (Fig. 19).	1332
nnitus aurium, due to stapedius paralysis. Low also, are painful to hear. No loss of taste. At rs.	Lesion of nerve above geniculate ganglion (928). (Fig. 36).	1333
rior two-thirds of tongue of same side.	Lesion of facial nerve between geniculate ganglion and stapedius branch. (Fig. 36).	1334
anterior two-thirds of tongue of same side.	Lesion of facial nerve between stapedius and chorda tympani branches. (Fig. 36).	1335
Tenderness near stylo-mastoid foramen.	Lesion of facial nerve below chorda tympani branch. (Fig. 36).	1336



CHART XXII c
Cerebro-Spinal Localization

Comprising Numbers 1298 and 1340 to 1346 on left side of Chart
and 1347 to 1369 on right margin.



CHART XXII d
Cerebro-Spinal Localization

Comprising Numbers 1299 on left side of chart
and 1372 to 1391 on right margin.



CHART XXII d
Cerebro-Spinal Localization

Comprising Numbers 1299 on left side of chart
and 1372 to 1391 on right margin.



CHART XXII e

Cerebro-Spinal Localization

TOPICAL DIAGNOSIS

LOCALIZATION OF LESION FROM ANALYSIS OF SYMPTOMS

MOTOR AND SENSORY PARALYSIS WITH EXAGGERATED REFLEXES JACKSONIAN EPILEPSY

DIAGNOSTIC SYMPTOMS AND TESTS

LOCALIZATION

1300 Both motor and sen- sory paraly- sis well marked. Reflexes present or exag- gerated, except in 1396.	Limited to both legs.	Paralysis severe. No ataxia. Organic reflexes much disordered. Some of the trunk reflexes are lost. Vertical extent of lesion is shown by the absence of the different trunk reflexes. Upper limit of lesion shown by the zone of hyperesthesia, limiting the anesthesia above.	Transverse lesion of spinal 1395 cord in dorsal region. (Myelitis.) (516-9, 829).
		Paralysis not so extreme. Marked ataxia. Loss of muscle sense. Organic reflexes not at all, or slightly, disordered. Trunk reflexes not abolished. Knee-jerks and other leg reflexes may be increased or abolished.	Lesion both in lateral and 1396 posterior columns of cord. (Ataxic Paraplegia.) (526, 660, 796) (Figs. 25-7.)
	Limited to both arms and both legs.	No involvement of cranial nerves. Priapism. Dyspnoea. Very dangerous, usually fatal.	Transverse lesion of spinal 1397 cord in cervical region. (512-5, 828) (Figs. 25-6).
		Involvement of some cranial nerves. Dysarthria and dysphagia. Very dangerous, usually fatal.	Lesion on both sides of 1398 brain stem (medulla, pons or crura cerebri, accord- ing to cranial nerves in- volved). (Figs. 19-21).
Spasmodic twitching of head and eyes to one side. Twitching may remain limited to these muscles or may extend to other muscles of face and neck and arm and later to leg of same side or may finally extend to muscles of both sides of body.			Lesion in or near base of 1399 middle frontal convolution of contralateral hemisphere. (Fig. 15).
Spasmodic twitching commences in one side of face. Twitching may remain limited to these muscles or may extend to others as above.			Lesion in or near lower 1400 quarter of the central con- volutions of contralateral hemisphere. (Fig. 15).
Spasmodic twitching in hand or arm. Twitching may remain limited to these muscles or may extend to face or to leg or to both simultaneously of same side and may later extend to muscles of other side of body also.			Lesion in or near middle 1401 half of the central con- volutions of contralateral hemisphere. (Fig. 15).
Spasmodic twitching of foot or leg. Twitching may remain limited to these muscles, or may extend to arm and later to face of same side and later to muscles of other side of body. (Figs. 15, 16).			Lesion in or near upper 1402 quarter of central convo- lutions or paracentral lob- ule of opposite hemisphere.
Spasmodic twitching, commencing simultaneously, in arm and face of same side, which later extends to muscles of the leg of the same side and still later to muscles of the opposite side of the body.			Lesion near and equally 1403 distant from motor area of face and arm in contra- lateral hemisphere. (Fig. 15).
Spasmodic twitching commencing in arm and leg of same side, which may later extend to face of same side and may later extend to muscles of the other side of body.			Lesion near and equally 1404 distant from motor area of arm and leg in contra- lateral hemisphere. (Fig. 15).
Spasmodic twitching commencing in face and arm and leg of same side, which may later extend to muscles of opposite side.			Lesion in inferior parietal 1405 lobule of contralateral hemisphere. (Fig. 15).

PLATES

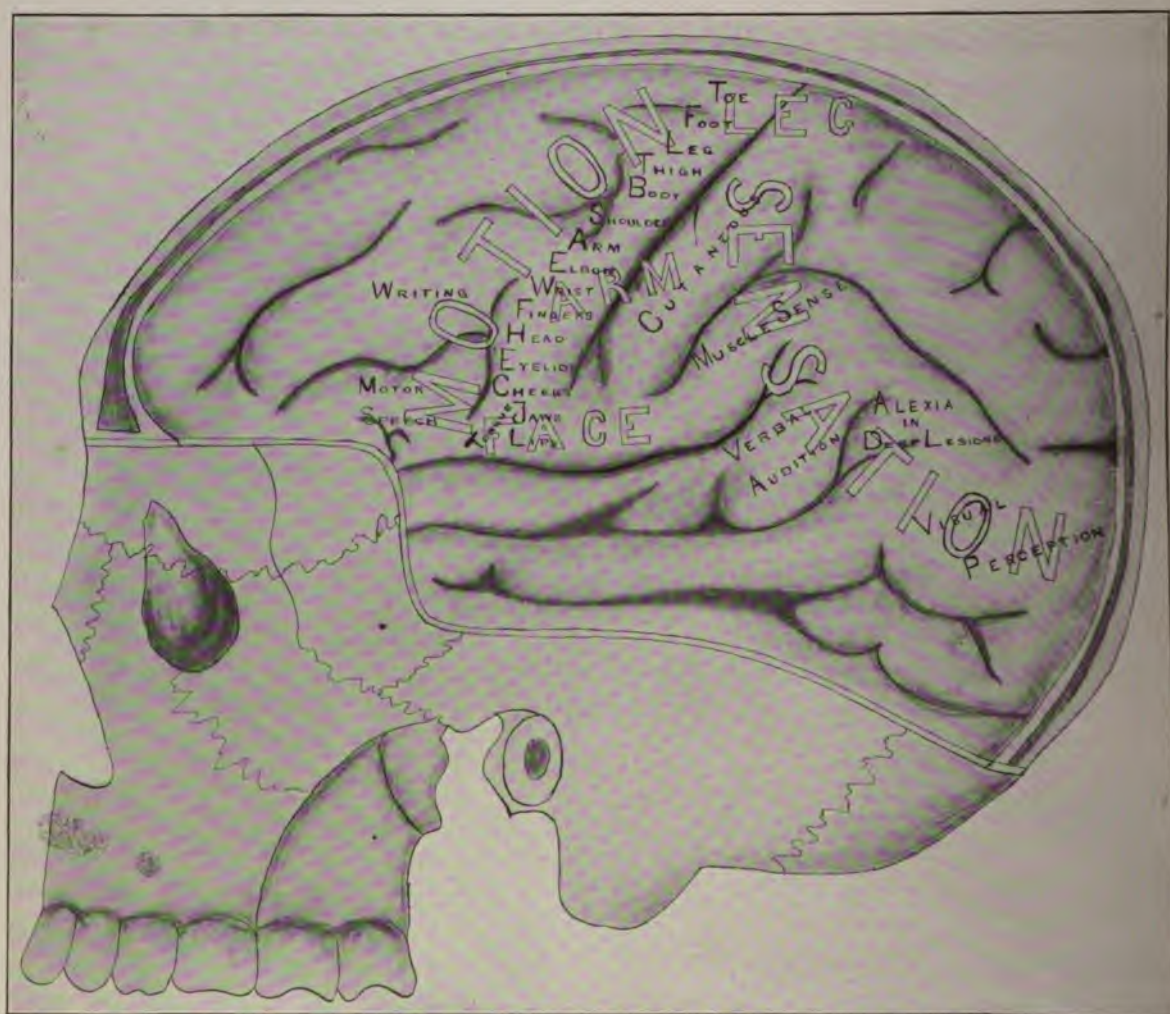


Fig. 15

Schematic representation of the convex surface of the left cerebral hemisphere, showing the motor and sensory areas, and the location of the cortical functions.

See 1282-6, 1348-9, 1352, 1355, 1362, 1367-9, 1373, 1376, 1379, 1380-1, 1387, 1389-91, 1400-5.

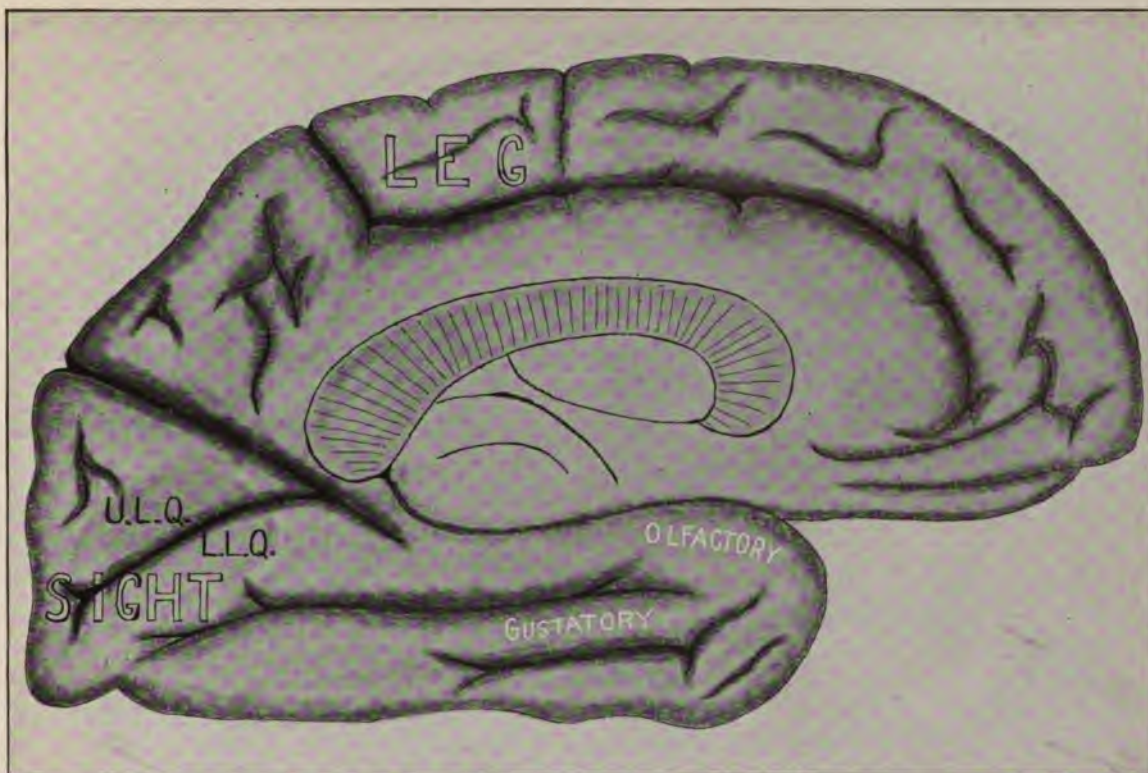


FIG. 16

Schematic representation of the median surface of the left cerebral hemisphere. U. L. Q.=Upper left quadrant of retina. L. L. Q.=Lower left quadrant of retina.
See 852-3, 856, 1285, 1364-6, 1402.

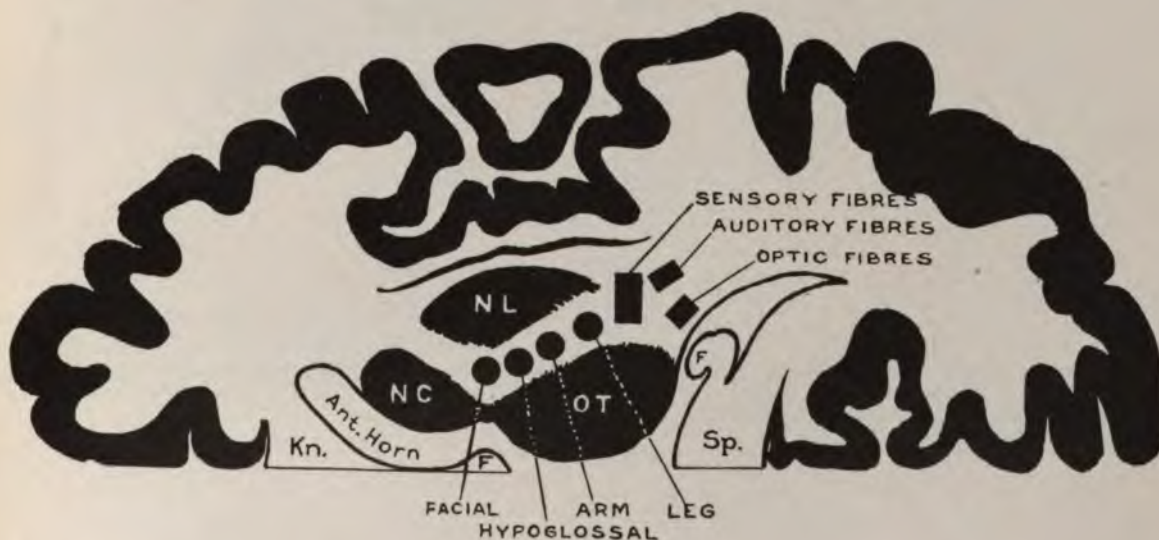


FIG. 17

Horizontal Section through Right Hemisphere showing the principal tracts situated in the Internal Capsule; Kn, Genu of Corpus Callosum; F, Fornix; NC, Caudate Nucleus; NL, Lenticular Nucleus; OT, Optic Thalamus. Sp, Splenium of Corpus Callosum.

See 1275-6, 1354, 1361, 1385-6.

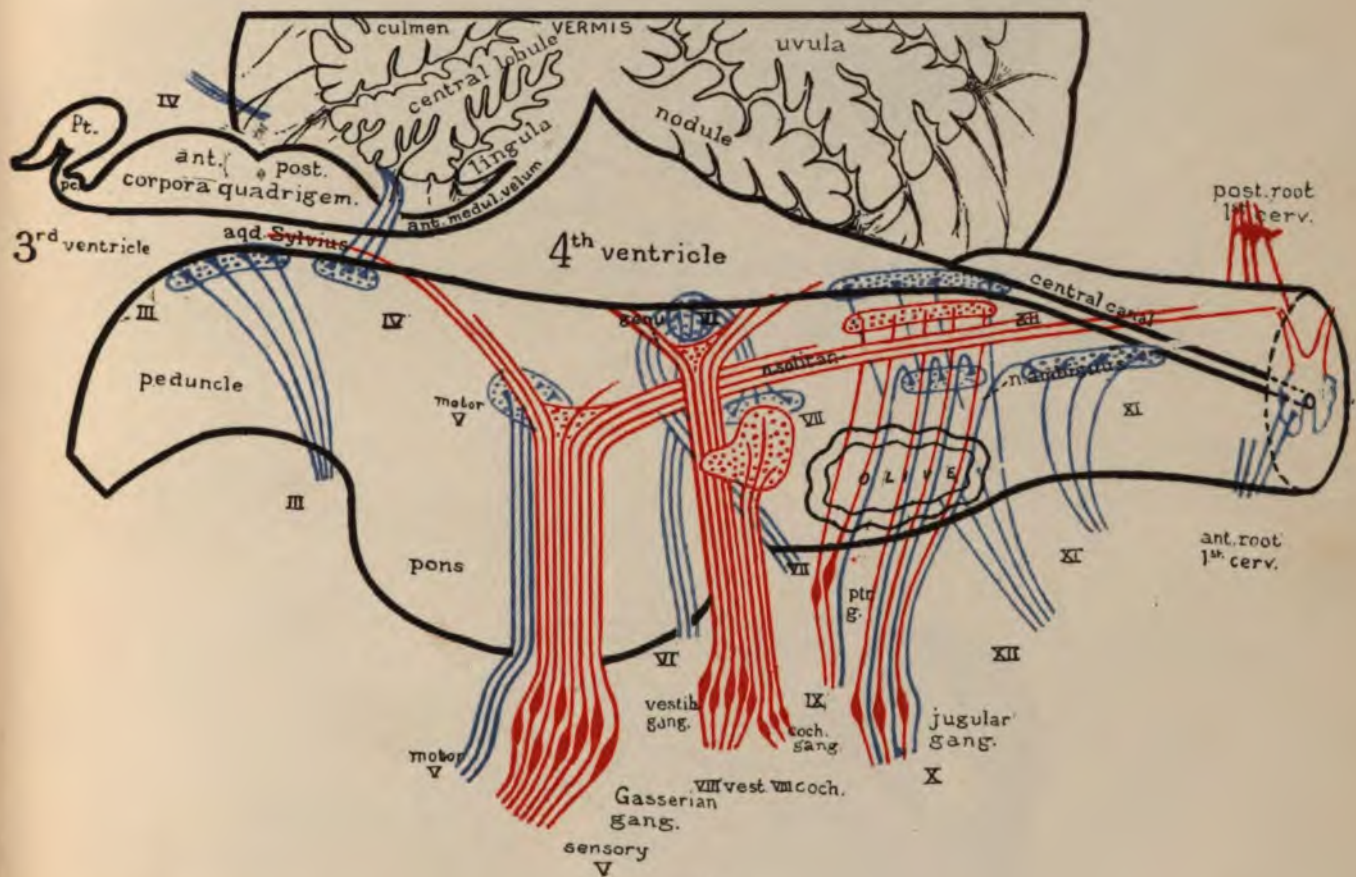


FIG. 19

Schematic representation of brain stem; showing nuclei and nerve roots.
The sensory nuclei and nerve roots are colored red, the motor blue.
See 1301-4, 1323-32, 1353, 1375, 1378, 1398.

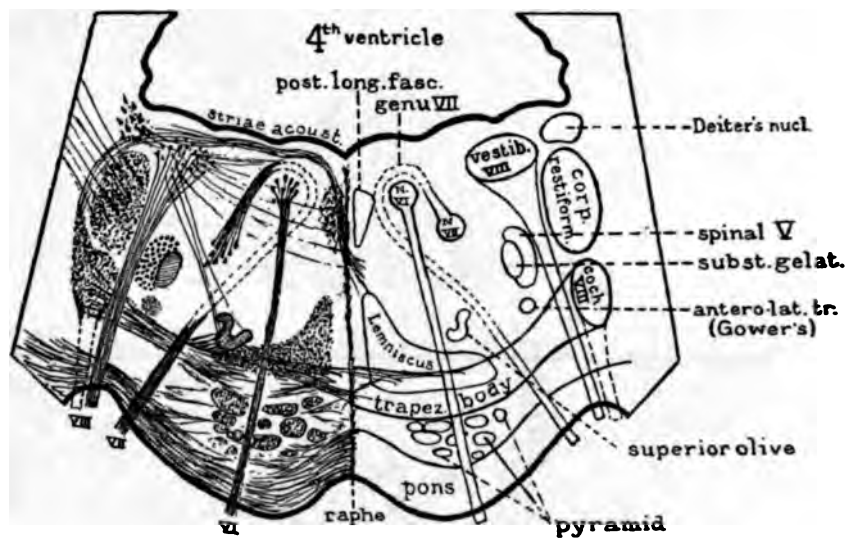


FIG. 20

Diagrammatic transverse section through the pons at a level slightly posterior to the superficial origin of the trigeminus.

See 1269, 1301-4, 1327-30, 1353, 1383, 1388, 1398.

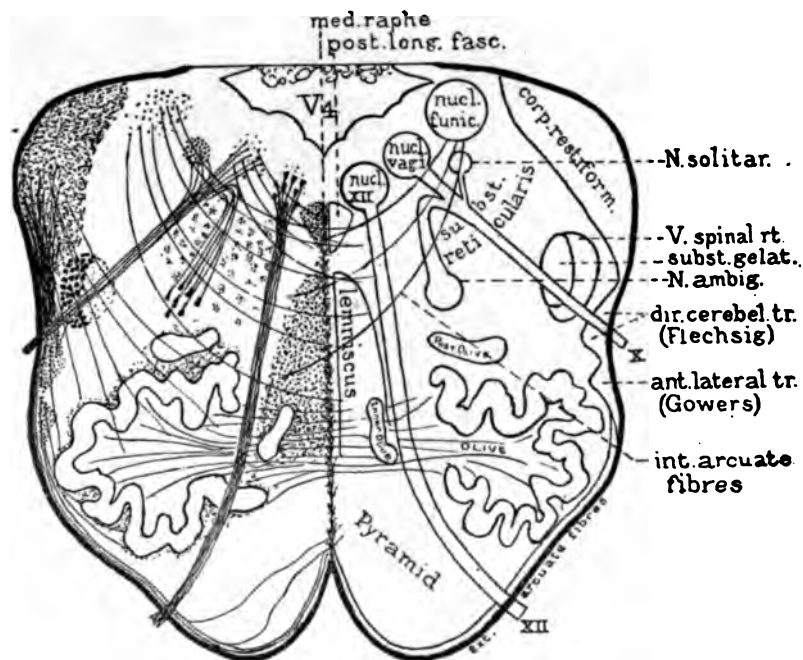


FIG. 21

Diagrammatic transverse section through the medulla, approximately near its middle.

See 1268, 1301-4, 1382, 1388, 1398.

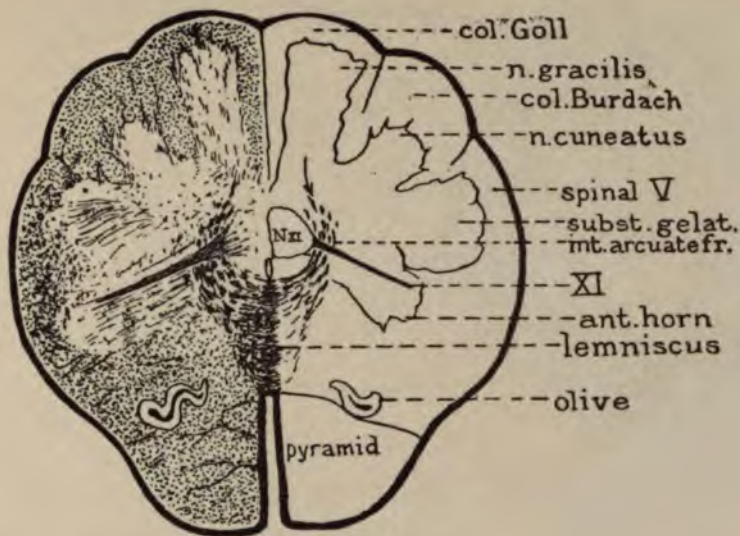


FIG. 22

Transverse section of medulla just above motor decussation and just above line of junction with the cord, showing the sensory decussation and the topography of the lowest level of the medulla.

See 1268

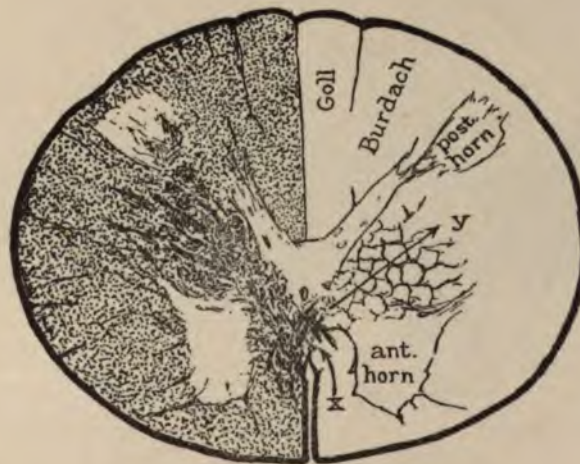


FIG. 23

Transverse section of the cord just at the line of junction with the medulla, showing the motor decussation and the topography of the uppermost level of the cord.

See 1268

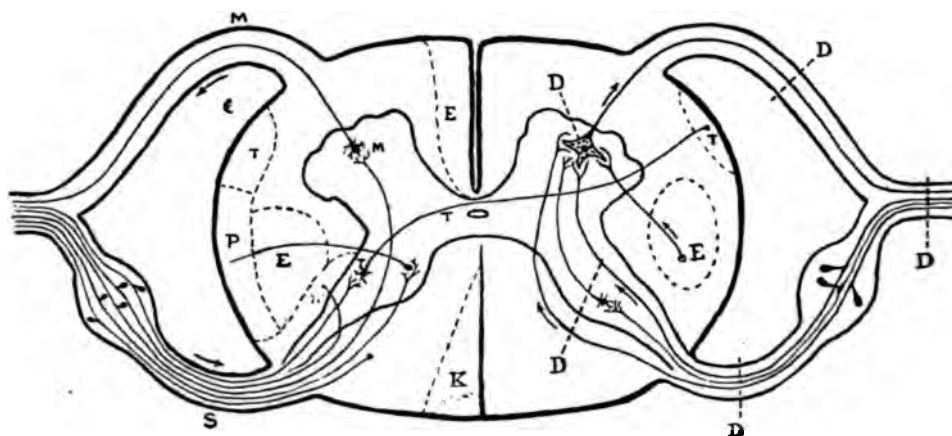


FIG. 24

DIAGRAMMATIC SECTION OF THE SPINAL CORD TO ILLUSTRATE ITS PHYSIOLOGY

Left side shows situation of lesions causing disorders of motion and sensation.

Right side shows situation of lesions causing disorders of reflex activity.

Destructive lesions at M or E cause diminution, slight irritative lesions, exaggeration, of motion. Destructive lesions at S cause permanent anaesthesia, analgesia, thermic anaesthesia and loss of muscle sense. Destructive lesions at T cause analgesia and thermic anaesthesia. Destructive lesions at P cause ataxia. Destructive lesions at K cause loss of muscle sense, ataxia and anaesthesia. Irritative lesions at S, K, T, or P, may cause exaggeration, or perversion, or both, of sensation. Destructive lesions at D cause diminution, and at E, exaggeration, of reflex activity. Slight irritative lesions at D cause exaggeration, and at E diminution, of reflex activity.

Symptoms of lesions at M are described in 252, 263, 495, 547, 789; 1148-9, 1233 and 1304 at E in 251, 254, 256, 525-6, 796-7 and 1212, 1372-4-7; at S in 824; at T in 1356-8-60; at P in 281, 654; at K in 280, 654a, 785, 1302, 1347 and 1350-1, 1396. The results of lesions at D and E are discussed in Chart V a.

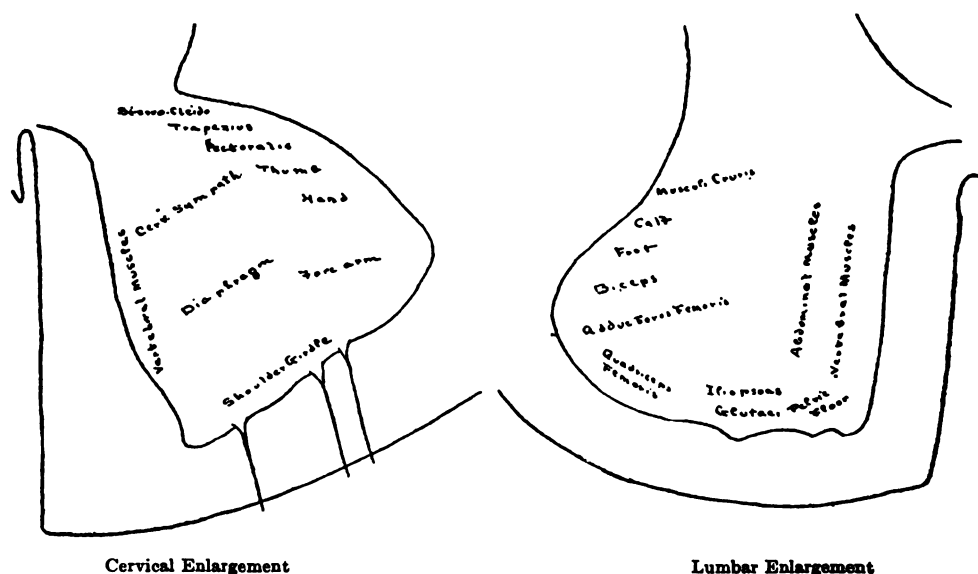


Fig. 25

LOCALIZATION OF NUCLEI IN THE ANTERIOR HORNS OF THE SPINAL CORD

(After Edinger modified from Sano.)

The Daily Dozen

RADIO HEALTH DRILL

as broadcast over

K P O

Hale Bros. and The Chronicle
Directed by the Y. M. C. A.



WILEY WINSOR, *Executive Secretary*
Director



CLAUDE B. EDWARDS
Accompanist

Meet these two men every morning
For fifteen minutes work and play
And you'll start your day correctly,
In a healthful, happy way.



J. K. HENDERSON
DIRECTOR OF
PHYSICAL ACTIVITIES

FAY D. BOWMAN
ASSOCIATE
BOYS PHYSICAL WORK

JOHN HART
ASSOCIATE
MENS PHYSICAL WORK

LLOYD WHITNEY
ASSOCIATE
SWIMMING ACTIVITIES


These expert Physical Directors of the Young Men's Christian Association of San Francisco are at your service for consultation regarding your physical needs.

This service for men and boys will be rendered free of charge. We welcome the opportunity to consult with parents regarding the problems of their boys.

Y. M. C. A. OF SAN FRANCISCO
220 Golden Gate Ave., corner Leavenworth St.

Phone Franklin 488

CHART OF POSITIONS

Post this Chart on a level with your eyes in the room where you are going to 

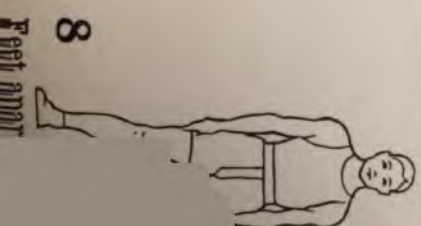
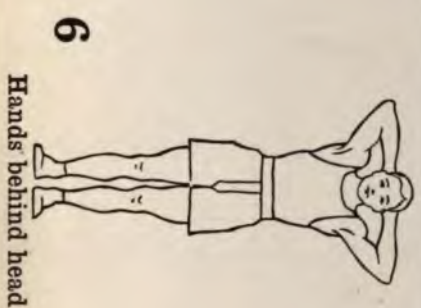
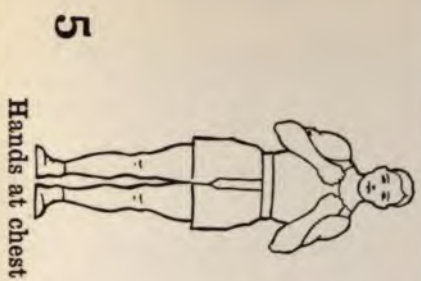
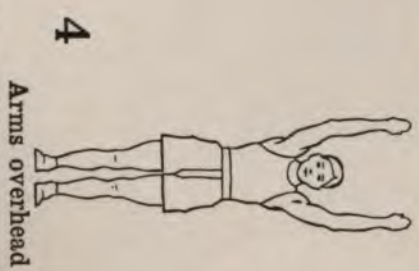
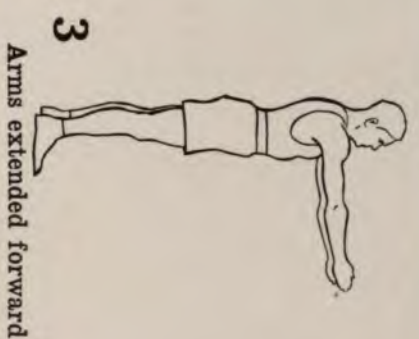
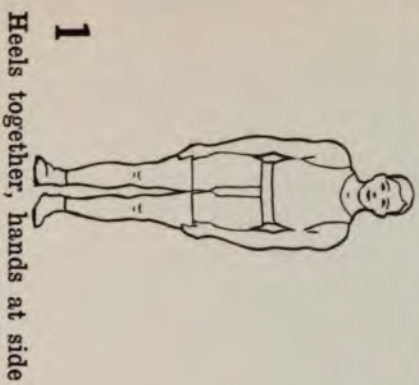
Used in Radio Health Drill Broadcast by

K P O

HALE BROS. and the CHRONICLE

Directed by the **Y.M.C.A.** San Francisco

make "A Fifteen Minute per Day Investment to keep Physically Fit"



PART III

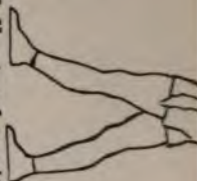
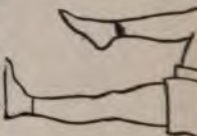







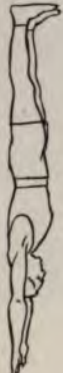


LOCALIZATION

OF

LESIONS WITHIN THE NERVOUS SYSTEM

BY

A CONSIDERATION OF THE
PARALYTIC AND IRRITATIVE SYMPTOMS
RESULTING FROM THEM

<p>9</p>  <p>Walk stand position</p>	<p>10</p>  <p>Knee raised position</p>	<p>11</p>  <p>Charge position</p>	<p>12</p>  <p>Full knee bend position</p>
<p>13</p>  <p>Forward body bend</p>	<p>14</p>  <p>Body rotation</p>	<p>15</p>  <p>Forward bend, body rotate</p>	<p>16</p>  <p>Front leaning rest</p>
<p>17</p>  <p>Back leaning rest</p>	<p>18</p>  <p>Lying on back</p>	<p>19</p>  <p>Sitting position</p>	<p>20</p>  <p>Inhale and exhale position</p>

(Copyright applied for by Y. M. C. A. of San Francisco, 1925)

Every morning, except Sunday, a fifteen minute Health Drill, based on the positions shown above, will be broadcast by the Department of Physical Education of the Y. M. C. A. of San Francisco over KPO.


Set the alarm clock to awaken you five minutes before KPO's Health Drill begins. Drink a glass of water, take the exercises, eat breakfast and get off to "A PERFECT START FOR A PERFECT DAY"

CHART OF POSITIONS

Used in Radio Health Drill Broadcast by

K P O

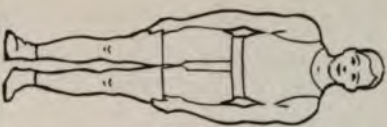
HALE BROS. and the CHRONICLE

Post this Chart on a level with your eyes in the room where you are going to 

Directed by the **Y.M.C.A.** San Francisco

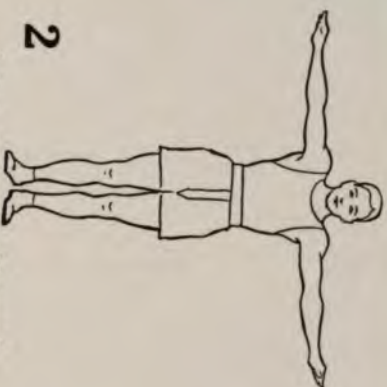
make "A Fifties Minute per Day Investment to keep Physically Fit"

1



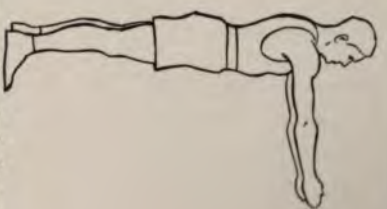
Heels together, hands at side

2



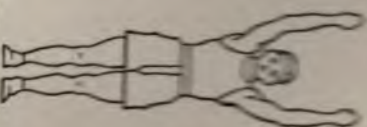
Arms extended sideward

3



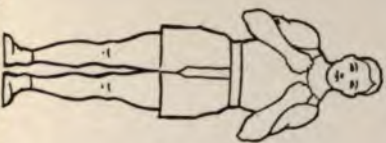
Arms extended forward

4



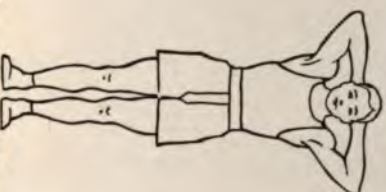
Arms overhead

5



Hands at chest

6



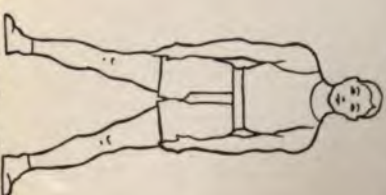
Hands behind head

7



Hands on hips

8



Feet apart, hands at sides

Healthy Men Rule The World

Good health is perhaps the most important, and at the same time, most desirable asset that any man, woman or child can have. Therefore, one of the important phases of the activities of the Young Men's Christian Association is its health building program. All exercises are worked out on a scientific basis and given in series, which if followed out carefully, will tend to build up health.

This chart has been prepared by the Physical Education Department of the Y. M. C. A. to be used in connection with the exercises broadcast over Station KPO, San Francisco.

These exercises are the same as are given in the gymnasium classes to our 10,055 members in San Francisco and will develop the whole body and keep anyone in good condition. However, fresh air, wholesome food, sufficient sleep and satisfactory working conditions are essential along with exercise. These exercises should be done with as little clothing on as possible, the room should be well ventilated; not too cold.

When these exercises are given in our gymnasium classes, we recommend that they be followed by a shower bath and a quick, brisk swim in the salt water pool. After exercising at home, a luke-warm sponge or shower bath, or a dry rub-down with a coarse bath towel should be taken as soon as the exercises are over.

Years of training have qualified our Staff to examine and properly prescribe a program of physical training. The courses are the result of 81 years of experience, checked nationally and internationally, with a membership which now exceeds 2,000,000. A man is advised to do only the things that are best for him and that will help him. Our Director of Physical Education will be glad to consult with you personally, or answer inquiries by mail. (Mail inquiries should enclose a self-addressed stamped return envelope).

The Radio Health Drill is directed by the Y. M. C. A. of San Francisco as a public service for which no remuneration is received or desired.

Eighteen Rules of Hygiene

1. Ventilate every room you occupy.
2. Wear loose, porous clothing suited to season, weather and occupation.
3. If you are an indoor worker, be sure to get recreation outdoors.
4. Sleep in fresh air always, in the open if you can.
5. Hold a handkerchief before your mouth and nose when you cough or sneeze and insist that others do so too.
6. Always wash your hands before eating.
7. Do not overeat. This applies especially to meat and eggs.
8. Eat some hard and some bulky foods; some fruits.
9. Eat slowly, chew thoroughly.
10. Drink sufficient water daily.
11. Have bowels act frequently, thoroughly, regularly.
12. Stand, sit and walk erect.
13. Do not allow poisons and infections to enter the body.
14. Keep the teeth, gums and tongue clean.
15. Work, play, rest and sleep in moderation.
16. Keep serene. Worry is the foe of health. Cultivate the companionship of your fellowmen.
17. Avoid self-drugging. Beware the plausible humbug of the patent-medicine faker.
18. Have your doctor examine you carefully once a year. Also consult your dentist at regular intervals.

Recommended by the
United States Public Health Service

Tables of Average Heights and Weights

MEN										
Age	15	20	25	30	35	40	45	50	55	
5 ft. 0 in.	107	117	122	126	128	131	133	134	135	
5 ft. 1 in.	109	119	124	128	130	133	135	136	137	
5 ft. 2 in.	112	122	126	130	132	135	137	138	139	
5 ft. 3 in.	115	125	129	133	135	138	140	141	142	
5 ft. 4 in.	118	128	133	136	138	141	143	144	145	
5 ft. 5 in.	122	132	137	140	142	145	147	148	149	
5 ft. 6 in.	126	136	141	144	146	149	151	152	153	
5 ft. 7 in.	130	140	145	148	150	153	155	156	158	
5 ft. 8 in.	134	144	149	152	155	158	160	161	163	
5 ft. 9 in.	138	148	153	156	160	163	165	166	168	
5 ft. 10 in.	142	152	157	161	165	168	170	171	173	
5 ft. 11 in.	147	156	162	166	170	174	176	177	178	
6 ft. 0 in.	152	161	167	172	176	180	182	183	184	
6 ft. 1 in.	157	166	173	178	182	186	188	190	191	
6 ft. 2 in.	162	171	179	184	189	193	195	197	198	
6 ft. 3 in.	167	176	184	190	195	200	202	204	206	
6 ft. 4 in.	172	181	189	196	201	206	209	211	212	
6 ft. 5 in.	177	186	194	201	207	212	215	217	219	
WOMEN										
Age	15	20	25	30	35	40	45	50	55	
4 ft. 8 in.	101	106	109	112	115	119	122	125	126	
4 ft. 9 in.	103	108	111	114	117	121	124	127	127	
4 ft. 10 in.	105	110	113	116	119	123	126	129	129	
4 ft. 11 in.	106	112	115	118	121	125	128	131	131	
5 ft. 0 in.	107	114	117	120	123	127	130	133	133	
5 ft. 1 in.	109	116	119	122	125	129	132	135	135	
5 ft. 2 in.	112	119	121	124	127	132	135	138	138	
5 ft. 3 in.	115	122	124	127	130	135	138	141	141	
5 ft. 4 in.	118	125	128	131	134	138	141	144	144	
5 ft. 5 in.	122	128	131	134	138	142	145	148	148	
5 ft. 6 in.	126	132	135	138	142	146	149	152	153	
5 ft. 7 in.	130	136	139	142	146	150	153	156	158	
5 ft. 8 in.	134	140	143	146	150	154	157	161	163	
5 ft. 9 in.	138	143	147	150	154	158	161	165	167	
5 ft. 10 in.	142	147	151	154	157	161	164	169	171	
5 ft. 11 in.	147	151	154	157	160	164	168	173	174	
6 ft. 0 in.	152	156	158	161	163	167	171	176	177	

For advice and information, write or phone to
MR. J. K. HENDERSON, *Director of Physical Activities*
Y. M. C. A., 220 Golden Gate Ave., San Francisco. Phone Franklin 461

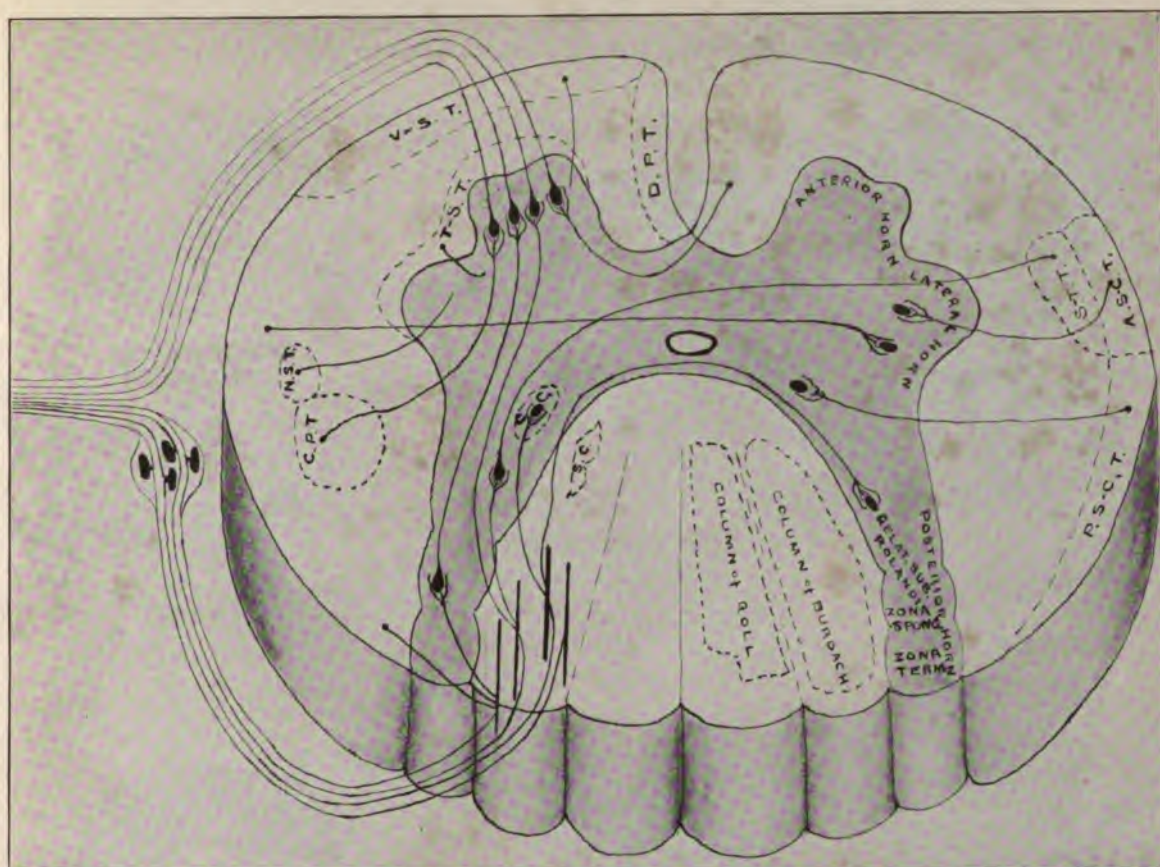


FIG. 26

A SCHEMATIC REPRESENTATION OF A TRANSVERSE SECTION OF THE SPINAL CORD; SEVERAL LEVELS BEING COMBINED INTO ONE

DESCENDING TRACTS

V.S.T.=vestibulo-spinal tract
T.S.T.=tecto-spinal tract
D.P.T.=direct pyramidal tract } cortico-spinal tract
C.P.T.=crossed pyramidal tract }
N.S.T.=rubro-spinal and thalamo-spinal tracts
S.C.=Schultze's comma

ASCENDING TRACTS

S.T.T.=spino-thalamic tract } (Gowers' tract)
A.S.C.T.=anterior spino-cerebellar tract }
P.S.C.T.=posterior spino-cerebellar tract (Flechsigs tract)
C.C.=Clark's column

On the left side of the cord are represented the nerve roots and those bundles of long fibers in the white columns which carry impulses downward from the brain to the spinal cord, and on the right side are represented those bundles of long fibers in the white columns which carry impulses upward from the spinal cord or spinal ganglia to the brain. It hardly needs to be stated that, although in this figure these long bundles of fibers are represented on one side only, they are really situated symmetrically on each side of the cord. The short fibers which connect different levels of the cord together are not represented in the figure.

Lesions involving the pyramidal tract give rise to a spastic paralysis described under 251, 254, 525-6, 796-7, 1212 and 1372-4-7. Lesions involving the anterior horns give rise to atrophic paralysis, the acute forms of which are described under 495, 789, 1148, 1233 and 1304; while the chronic forms are described under 547, 695, 1149 and 1304. Lesions involving the posterior horn give rise to symptoms described under 1302. Lesions of posterior columns give rise to symptoms described under 785, 1302, 1347, 1350-1 and 1396. Lesions of the spino-cerebellar tract give rise to symptoms described under 281 and 654. Lesions of the spino-thalamic tract and of the anterior commissure of the gray matter give rise to symptoms described under 365, 811 and 1356-60. Lesions of the whole of one lateral half of the cord give rise to symptoms described under 442, 509, 840 and 981; while lesions of the whole transverse section of the cord give rise to symptoms described under 485, 513-4, 517-8, 520, 549-50, 791, 795, 825, 828-9, 835 and 980, 1148a, 1309-10, 1395-7.

FIG. 27

Schematic representation of the more important diseases of the spinal cord.

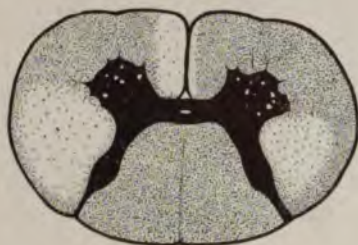


Locomotor Ataxia
(lumbar region)

See 345, 416, 419-20, 433, 661, 756,
784, 827, 891, 894, 911, 979, 987,
1004, 1015, 1172, 1186, 1217 and
1231



Locomotor Ataxia
(cervical region)



Amyotrophic Lateral Sclerosis

See 547, 695, 797, 1149;
and 525, 670 and 797



Descending Degeneration of
Pyramidal Tracts



Acute Stage Chronic Stage
Anterior Poliomyelitis
See 416, 419, 495, 789 1148
and 1233



Syringomyelia
See 552, 693, 837-9, 1009, 1150a
1170, 1187, 1357 and 1359



Compression Myelitis with the conse-
quent Ascending and Descending
Degenerations. See 520, 795.

No. 3 shows the point of the compression with
the whole transverse section of the cord the
seat of an inflamma

No. 1 shows ascending degeneration of the
columns of Goll, of the spino-thalamic tracts,
and of the anterior and posterior spino-cerebellar
tracts.

No. 2, close to the lesion, shows in addition a
slight degeneration of the columns of Burdach.

Nos. 4-6 show degeneration of the crossed
and direct pyramidal tracts of the vestibulo-
spinal, rubro-spinal, and thalamo-spinal tracts
and of Schultze's comma.

The upper series face up and the lower down.



Medulla cervicalis

SCHEMATIC REPRESENTATION OF SOME POINTS IN THE PHYSIOLOGY AND PATHOLOGY OF THE SPINAL CORD AND PERIPHERAL NERVES.

Fig. 28. Diagram to illustrate the mechanism of the bladder reflex

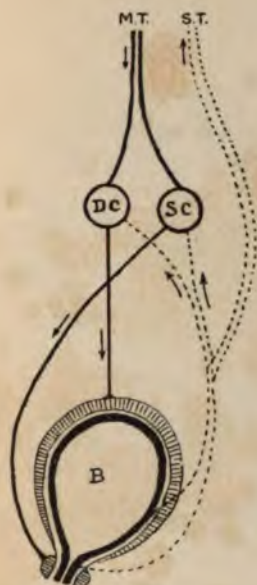


Fig. 28

B represents the bladder. SC represents the reflex centre, with its motor and sensory neurons, for the sphincter of the bladder, which is excited to action by urine in the neck of the bladder or in the prostatic urethra. DC represents the reflex centre, with its motor and sensory neurons, for the detrusor of the bladder, which is excited to action by the distention of the walls of the bladder. These two reflexes are antagonistic and the sensory surface irritated being much larger in the latter (DC), than in the former (SC), reflex, the detrusor reflex will eventually overpower the sphincter reflex under normal conditions. ST represents the sensory tract connecting the bladder with the brain, by means of which the individual is informed as to the degree of fullness of the bladder. MT represents the motor tract connecting the cerebral with the spinal centre by means of which the individual can inhibit the activity of either centre (up to a certain degree) and increase the activity of the antagonistic centre.

FIG. 29 illustrates effects of lesions of cauda equina.

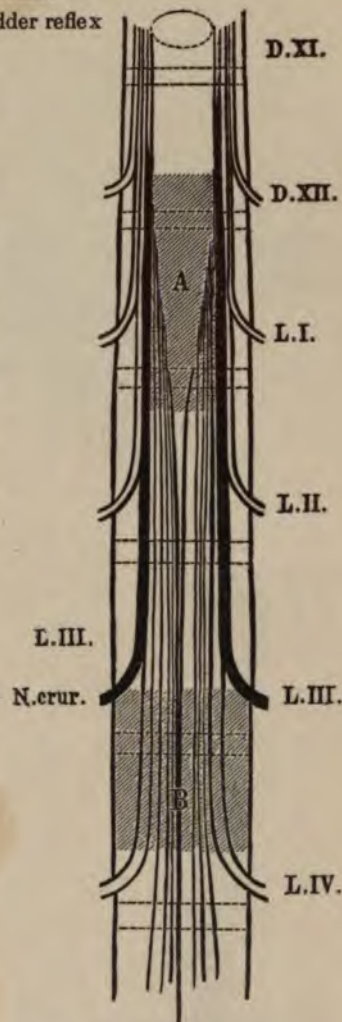
If the lesion is at "A" there is complete motor paralysis of both legs, and complete anesthesia of the whole of both legs and of the perineum, buttocks, scrotum and penis, and all reflexes of the legs are abolished.

If the lesion is at "B" there is complete motor paralysis of both legs, except the flexors of the thigh and the extensors of the leg, and complete anesthesia of the perineum, buttocks, scrotum and penis, and of the posterior surface of the thighs, the posterior and lateral surfaces of the legs, and all of the foot, except a small area on its inner surface. All the reflexes of the legs except the knee-jerks are abolished.

In both cases the muscles atrophy, there is no zone of hyperesthesia above the anesthesia and the bladder and rectum show a motor and sensory paralysis.

If the lesion is limited to the conus medullaris there is a paralysis of the rectum and bladder and an anesthesia of the penis, scrotum, perineum, one inch about anus, and the upper two-thirds of the posterior surface of the thighs. Otherwise there is no paralysis of motion or sensation.

See 487, 721, 1007, 1308.



(After Fr. Schultze-Köster.

Fig. 29



Fig. 30

Showing the innervation of muscles through more than one nerve root, so that the destruction of one nerve root or of one group of nerve cells does not cause a complete and permanent paralysis.

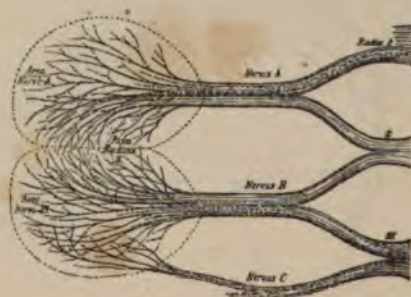


Fig. 31

A diagram showing that a given sensory area of the skin is supplied by filaments from several nerve roots; so that division of one root does not necessarily produce total anesthesia. It also shows the peripheral overlapping; so that the area supplied by one nerve can be almost completely supplied by neighboring nerves.

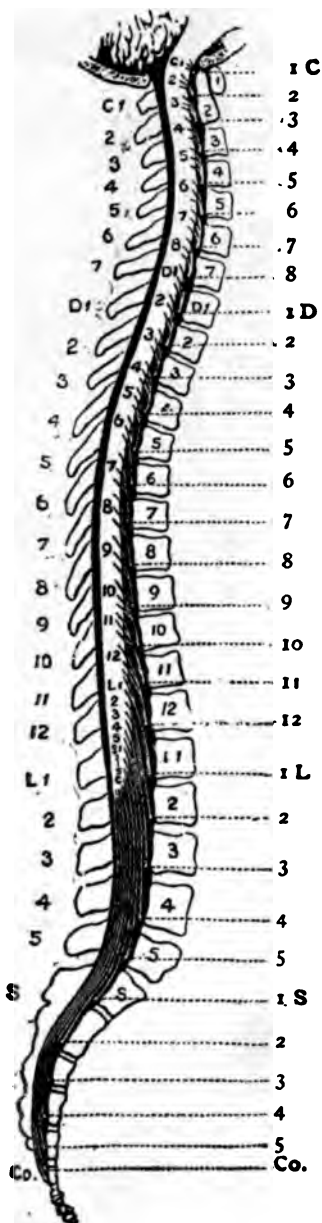


FIG. 32

MOTOR AND REFLEX FUNCTIONS OF THE SPINAL-CORD SEGMENTS (MODIFIED AFTER STARR AND EDINGER)

SEGMENT	MUSCLES	REFLEXES
Cervical	Sternomastoid	
2-3	Trapezius	
	Scaleni	
	Small rotators of head	
	Diaphragm	
4	Lev. ang. scap.	Dilatation of pupil by irritating side of neck, 4 cervical to 1 dorsal
	Rhomboids	
	Spinati	
	Deltoid	
	Supinat. long	Scapular reflexes, 5 C-1 D
5	Biceps	Supinat. long., 5 C
	Supinat. brev.	
	Serrat. mag.	
	Pectoralis (clav.)	Biceps, 5-6 C
	Teres minor	Triceps, 6 C
6	Pronators	Posterior wrist, 6-8 C
	Brachialis ant.	Scapulo-humeral, 7 C
	Triceps	
7	Long extensors of wrist and fingers	Anterior wrist, 7-8 C
	Pectoralis (costal)	
	Latiss. dorsi	Palmar, 7 C-1 D
	Teres maj.	
8	Long flexors, wrist and fingers	Epigastric, 4-7 D
	Extensors of thumb	
Dorsal 1	Intrinsic hand-muscles	Abdominal, 7-11 D
2-12	Dorsal and abdominal muscles	
Lumbar	Abdominal muscles	Cremaster, 1-3 L
1	Iliacus	
	Psoas	Patellar, 2-4 L
2	Sartorius	Bladder, 2-4 L
	Flexors of knee	
	Quad. femoris	
3	Int. rotators of thigh	
	Adductors of thigh	Rectal, 4 L-2 S
4	Abductors of thigh	
	Tibialis ant.	Gluteal, 4-5 L
	Calf-muscles	
5	Ex. rotators of thigh	
	Extensors of toes	
Sacral	Peronei	Achilles, } 1-3 S
1-2	Long flex. of toes	Ankle-clonus, }
	Intrinsic foot-muscles	Plantar, 1-2 S
3-5	Perineal muscles	Anal, } 3-5 S
		Virile, }

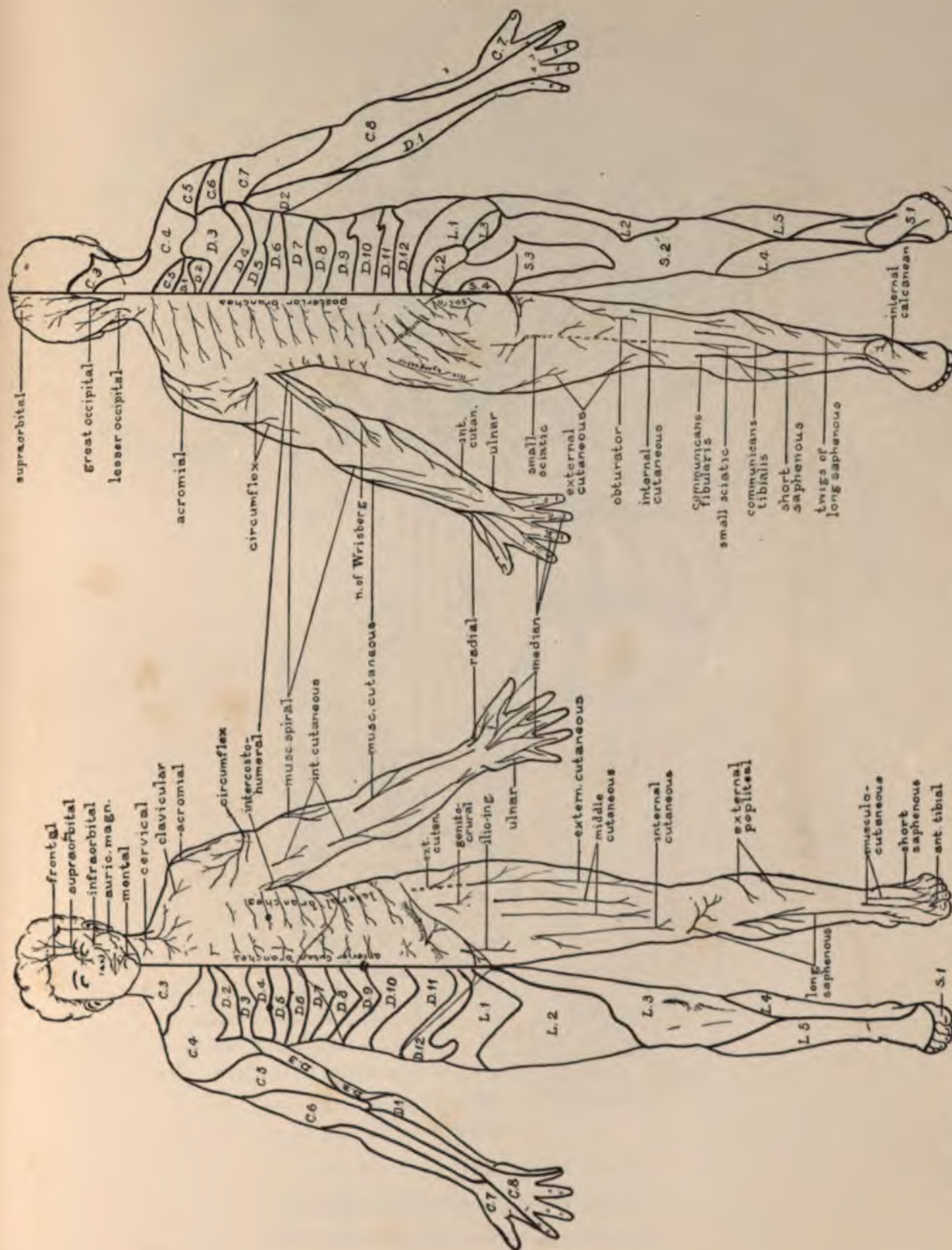


FIG. 33.—Representing on right side of body the sensory cutaneous areas connected with each spinal segment, and on the left side the cutaneous distribution of the sensory nerves. See 636, 822, 824 and 1301-4

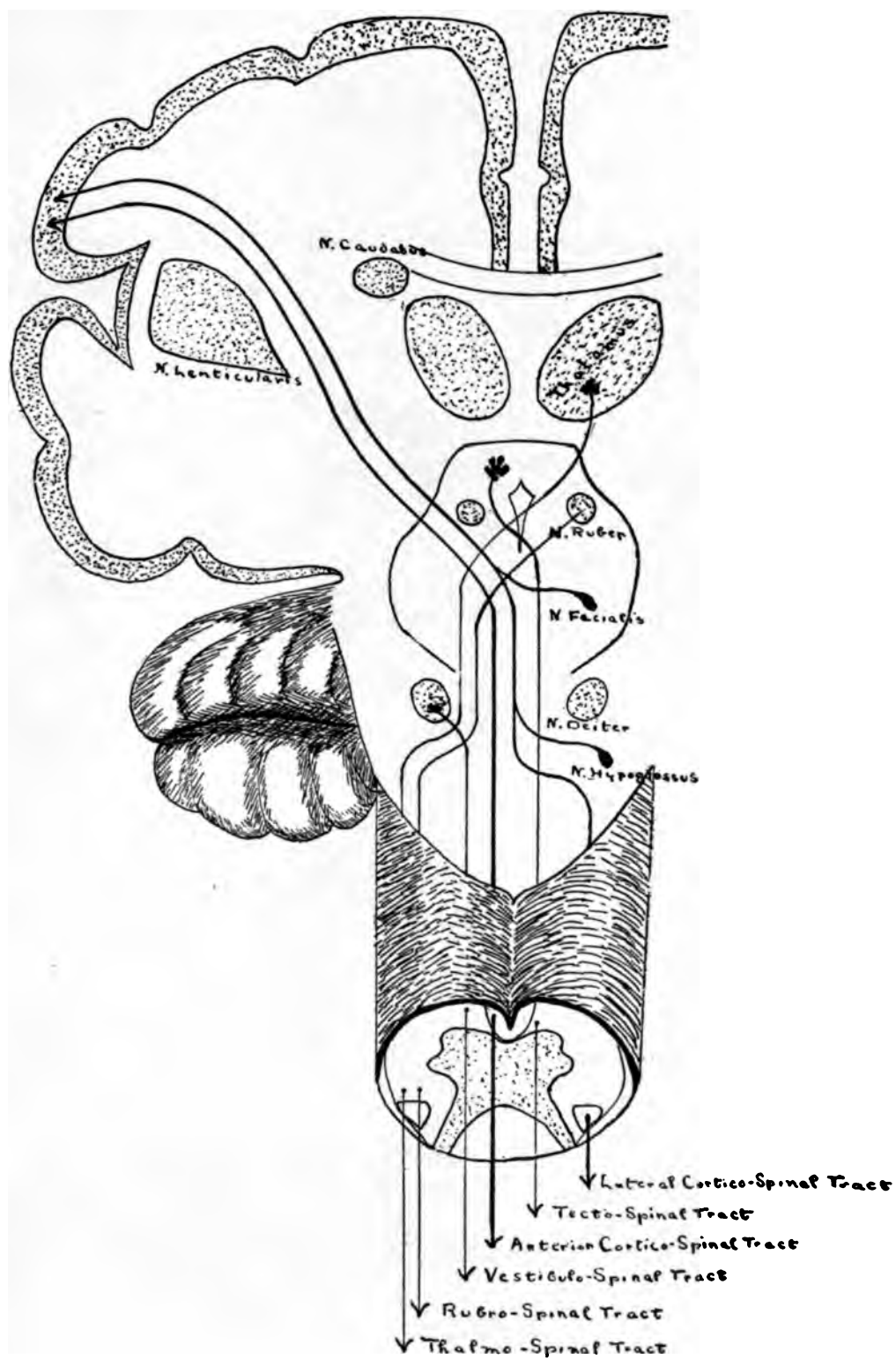


Fig. 34

LONG MOTOR PROJECTION TRACTS

For lesions involving these tracts see under Fig. 26.

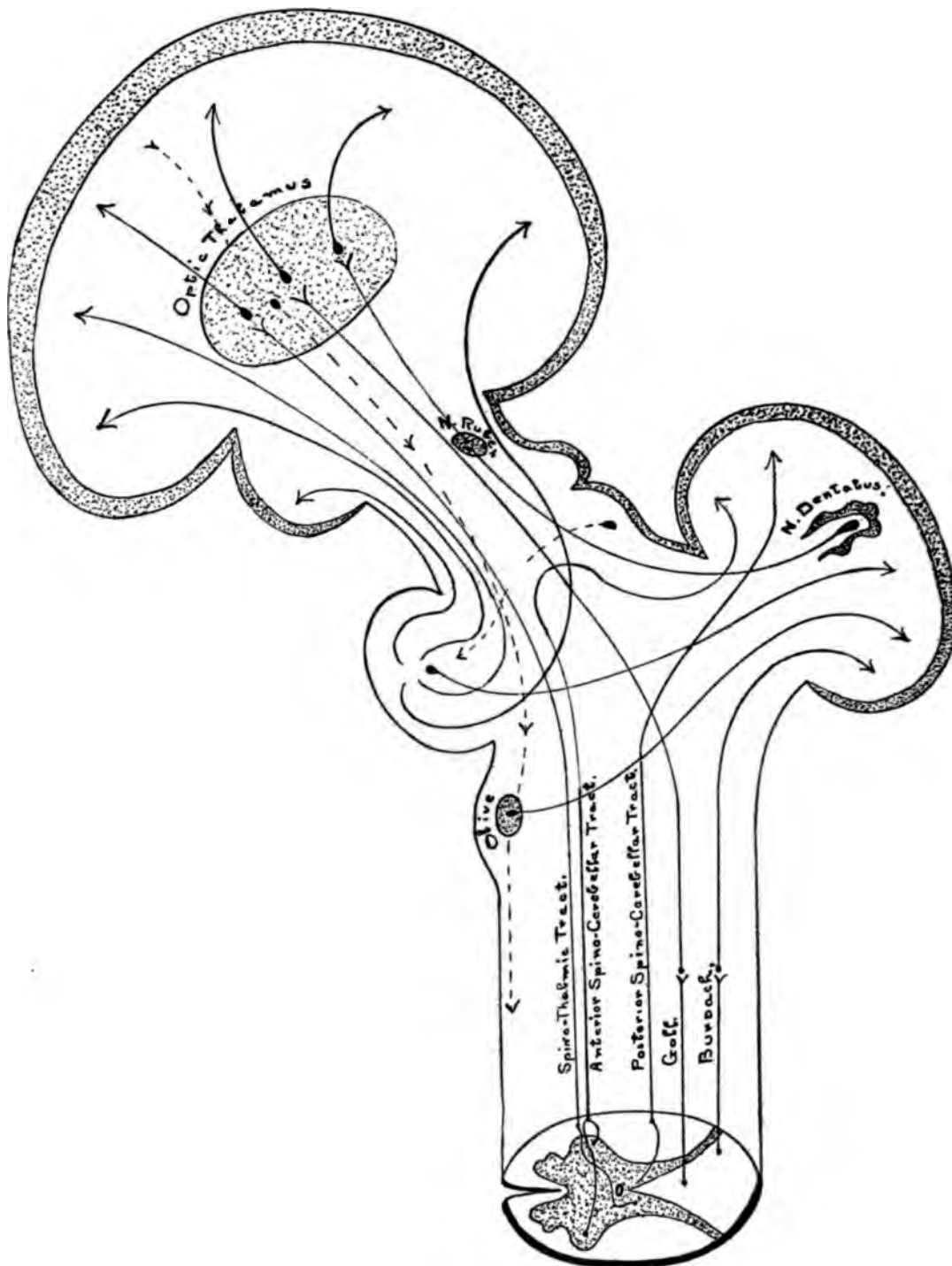


Fig. 35

LONG SENSORY PROJECTION TRACTS

For lesions involving these tracts see under Fig. 26.

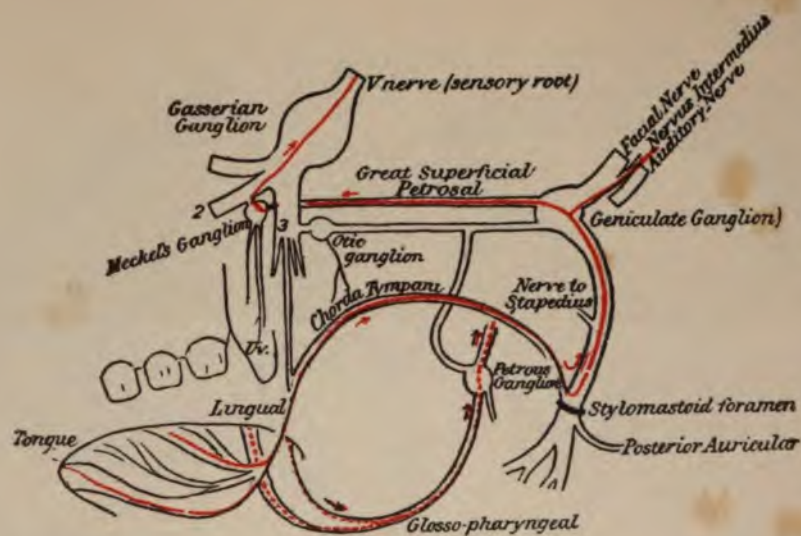


Fig. 36
 DIAGRAM OF TRIGEMINAL, FACIAL AND GLOSSO-PHARYNGEAL NERVES, SHOWING
 COURSE OF TASTE FIBRES.
 (After Purves Stewart)

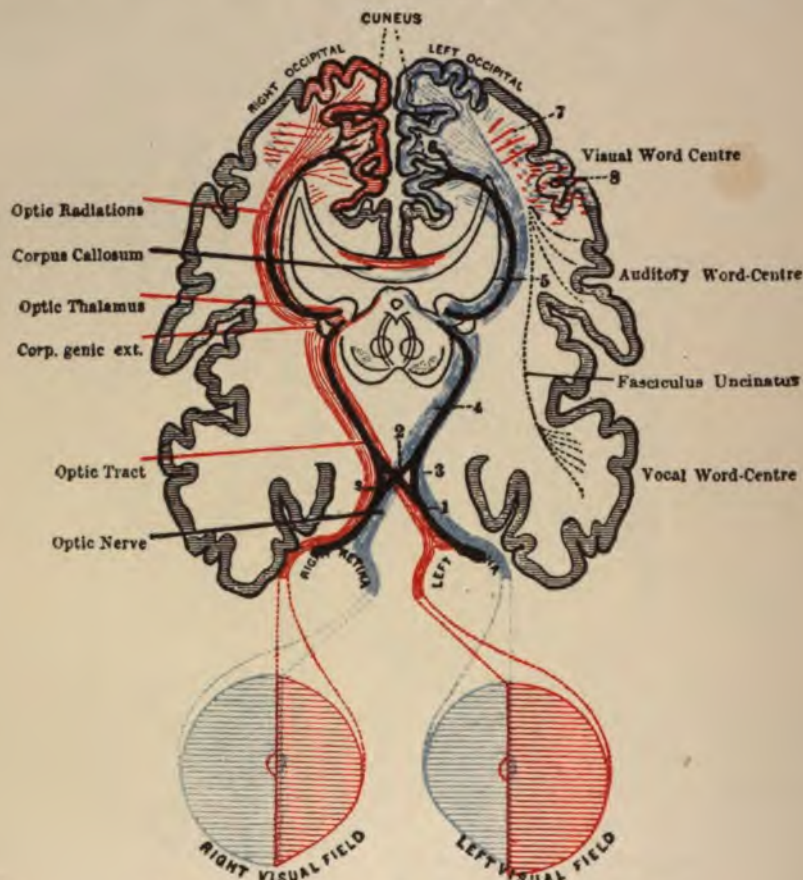


Fig. 37
 DIAGRAM ILLUSTRATING HEMIANOPIA
 (Modified from Viallet)

- | | |
|--|--|
| Lesion at 1 produces blindness of one eye. | Lesion at 5 produces R. hemianopia with normal pupil reaction. |
| Lesion at 2 produces bi-temporal hemianopia | Lesion at 6 produces R. hemianopia with normal pupil reaction. |
| Lesion at 3 produces bi-nasal hemianopia | Lesion at 7 produces psychic blindness. |
| Lesion at 4 produces R. hemianopia with hemiopic pupil reaction. | Lesion at 8 produces Alexia. |

The heavy black lines represent the fibers from the macula lutea in each retina, the point of central, or clearest vision.

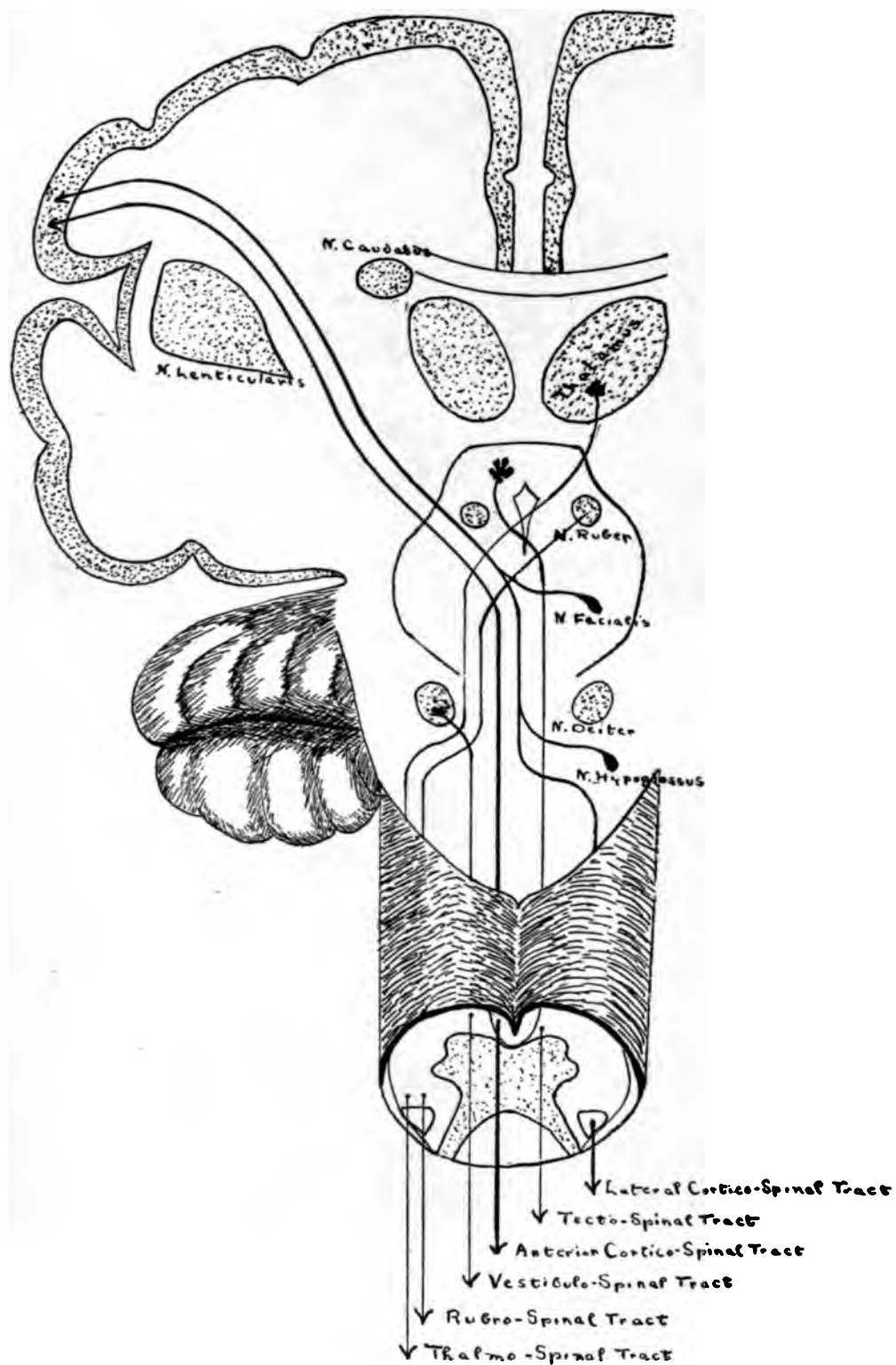


Fig. 34

LONG MOTOR PROJECTION TRACTS

For lesions involving these tracts see under Fig. 26.



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